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SYNOPSIS OF LECTURES

UPON

Diseases of the Nervous System

DELIVERED AT THE

COLLEGE OF PHYSICIANS AND SURGEONS, MEDICAL DEPARTMENT OF COLUMBIA UNIVERSITY, NEW YORK

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M. ALLEN STARR

SYNOPSIS OF LECTURES

UPON

DISEASES OF THE NERVOUS SYSTEM.

A KNOWLEDGE of the structure and functions of the central nervous system is an essential preliminary to the study of its diseases. The human nervous system is the most complex of all nervous structures, and is the culmination of the process of evolution. It will aid in the understanding of its structure if the various stages in its evolution are known.

THE EVOLUTION OF THE NERVOUS SYSTEM.

The simplest form of a nervous system consists of a single mass of gray matter with a sensory and a motor nerve.

The mass of gray matter receives impulses sent from the surface by the sensory fibre, acts in response to the impulse, and the action is transmitted to a motor mechanism by the motor nerve.

Example. The closure of an oyster-shell when the oyster feels any object touching its sensory surface. Parallel action in human system—any spinal reflex—*e.g.*, knee-jerk.

Several such single masses may be joined to one another, each with its sensory and motor nerve. The masses receive impulses in succession and act in succession.

Example. Wave-like motion of a jelly-fish or caterpillar.

A compound type of nervous system consists of several subordinate masses joined together and presided over by a single higher mass, which is joined to each of the lower masses. The higher mass receives impulses from the lower masses, not directly from the surface. It sends impulses to the motor mechanisms through the lower masses, not directly. It secures a certain definite succession and combination of action in the lower masses.

Examples. The nervous system of a frog experimentally deprived of its hemispheres, capable of automatic acts of swimming,

The automatic act of respiration in man. In man the facial, laryngeal, intercostal, thoracic, and diaphragmatic respiratory muscles act in definite succession and rhythm in the act of breathing; and this act is presided over by a single centre in the medulla controlling the lower medullary and spinal centres.

The highest type of nervous system is the complex type. There are the lower centres as in the simple type. There are higher centres controlling the lower centres, as in the compound type. There is a supreme mass controlling both the others.

This is the form found in all vertebrates, and the degree of evolution in the highest or supreme mass determines the place of the animal in the scale of intelligence.

In man the supreme mass is the cerebral cortex, the next lower or automatic centres are the optic thalami and basal ganglia, including gray masses in the cerebral axis; the lowest or reflex centres are the cranial nerve nuclei in the cerebral axis and the gray matter of the spinal-cord segments.

These distant gray masses are joined with one another in all possible combinations by means of the white nerve tracts, which pass in all directions around and within them.

Read Herbert Spencer, "Principles of Psychology," Part I., Chaps. I.–IV.
Charles Mercier, "The Nervous System and the Mind," Part I.

The Neuron.

The primary elements of the nervous system are neurons, and the nervous system from the simplest to the most complex in the order of evolution consists of a collection of such neurons of greater or lesser complexity.

The neuron consists of:

(1st) A cell body whose shape and size vary at different parts of the nervous system.

(2d) Dendrites or protoplasmic prolongations of the cell body which divide and subdivide as they extend outward from it much like the roots of a tree. The surface of these dendrites is rough, appearing like moss under the microscope on account of the presence of fine granules. Nervous impulses are collected by these dendrites and transmitted to the cell body. Fine filaments have been demonstrated passing from each dendrite through the cell body and entering the axon.

(3d) The axon or axis-cylinder process of the cell body is a single long, projecting fibre passing outward from the cell body and transmitting impulses from the cell body to a distance. The neuraxon gives off fine collateral branches in its course and terminates as do its collaterals in a fine brush-like end. This brush is supposed to come in contact with the gemmules of the dendrites of other neurons so that connection between different neurons is possible. The axon is made up of many fine fibrillæ which, as already stated, have come through the cell from the dendrites. The cell body controls nutrition of the dendrites and axon so that when it is destroyed they atrophy.

The Structure of a Neuron.—A fine trabeculum or framework appears to be filled with semifluid protoplasm, which protoplasm is used up by action of the neuron, thus leading to alterations in size in accordance with the amount of work performed. The normal cell body shows under staining with methyl blue a granular appearance, and these granules are termed chlorophyl bodies and appear to have a regular arrangement within the cell. The protoplasm within the trabeculæ does not stain. In a degenerated cell marked changes in the staining capacity and an irregularity of arrangement of the chlorophyl bodies are noticed.

Varieties of Neurons.—Neurons have been divided according to their function into: (1st) Centrifugal, which are either (*a*) motor, (*b*) secreting, (*c*) trophic. (2d) Centripetal, or sensory. (3d) Intrinsic, or association. Neurons have also been divided into a series according to their position, into (1) primary neurons whose cell body lies in the central nervous system or in the ganglia, and whose axon extends thence to some peripheral portion of the body, *e.g.*, to a muscle, to a gland, to the skin; (2) secondary neurons whose body lies in the central nervous organs and whose axon extends thence to some other part of the central nervous organs to terminate about a primary neuron. These neurons may lie in the cortex and brain or in the basal ganglia or medulla.

All simple reflex acts involve the transmission of impulses through primary neurons only.

All transmission of voluntary motor impulses, or conscious sensory impulses, involves the action of both primary and secondary neurons.

The Human Nervous System.

The human nervous system consists of a peripheral system and a central system.

The peripheral system comprises all the nerves.

The central system includes:

First, the spinal cord and cranial nerve nuclei.

Secondly, the cerebellum; gray masses of the medulla, pons, and crura cerebri; corpora quadrigemina, optic thalami, and corpora striata.

Thirdly, the cerebral cortex.

THE PERIPHERAL NERVES AND THEIR DISEASES.

STRUCTURE of the nerves. Bundles of nerve-fibres—large or small.

Structure of a nerve-fibre: 1. Axis cylinder, a long process of some nerve-cell. 2. Medullary sheath of myelin—a fatty insulator, interrupted at the nodes of Ranvier. 3. Connective-tissue sheath of Schwann, with its nucleus. Nerve-fibres are bound into bundles by connective tissue; endoneurium, perineurium. The visceral nerve-fibres have no medullary sheath.

All nerve-fibres branch during their course in the central nervous system, sending out "collaterals," but do not branch in their peripheral course. They arise from a nerve-cell. They terminate in fine brush-like expansions which are spread out in the skin, or on a muscle, or around another nerve-cell.

Nerves are divided into motor nerves and sensory nerves, according to the mechanism to which they are attached. Motor nerves convey impulses outward, sensory inward. Motor nerves grow outward from large nerve-cells in the spinal cord. Sensory nerves grow from cells in the posterior spinal ganglia, which send one branch to the surface, and the other into the spinal cord.

NEURITIS.

Neuritis may occur in a single nerve, or in a plexus.

Causes.—Injuries, strains, pressure, compression in fractures, rheumatism, gout, cold.

Pathology.—1. Parenchymatous neuritis, congestion and exudation in nerve-trunk; swelling and degeneration of individual fibres; (1) axis cylinder broken and disintegrated in fatty mass; (2) medullary substance separated into segments and fatty globules, and mingled with granules and nucleated cells; (3) connective-tissue sheath shrunken and empty. Nuclei multiply. Products of degeneration liquefy and are absorbed.

Regeneration occurs by growth of new fibres outward from the healthy stump into the old sheath.

2. Interstitial neuritis. The connective tissue of the endoneurium and perineurium increases in volume and the nerve-fibres are compressed by the congested vessels and by the new tissue. These forms usually occur together in non-traumatic cases.

Symptoms.—Weakness or paralysis with atrophy and reaction of degeneration in muscles supplied by nerves involved. Reaction of degeneration (R. D.) is a change in the action of the nerves and muscles to the electric currents. The response of the nerve to both currents is lost. The muscle loses its faradic reaction. The galvanic reaction is changed, the muscle responding more actively to the closure of the positive pole (anode) than to that of the negative pole (kathode). Numbness or anæsthesia in skin supplied by nerve involved. Vasomotor and trophic changes in the distribution of nerve involved. Coldness from sluggish metabolism; glossy skin; œdema; pain and tenderness at seat of inflammation in interstitial cases.

Course.—Slow spontaneous recovery when continuity of nerve is preserved. Otherwise, no recovery.

Treatment.—If nerve is broken unite divided ends. Rest the part injured but exercise the muscles paralyzed by electricity and massage. Use hot applications, sedative lotions. Protect from cold by cotton batting. Morphine for pain.

Multiple Neuritis.

Multiple neuritis is an inflammatory or degenerative disease of the peripheral nervous system varying in extent and intensity and affecting symmetrical parts of the body.

The motor nerves may be involved alone (*e.g.*, in lead and mercury poisoning), or the sensory nerves alone (*e.g.*, in coal-gas poisoning), or both may be involved together (*e.g.*, in alcoholic neuritis). The distal parts of the nerves are commonly affected to a greater extent than the central parts. Hence the symptoms appear chiefly in the distal parts of the extremities.

The pathological process is the same as in simple neuritis and may be parenchymatous alone, or interstitial alone, or both.

Etiology.—1. Toxic cases: Alcohol, lead, arsenic, bisulphide of carbon, copper, zinc, mercury, phosphorus, coal gas.

Both arms or both legs are more often affected than a single limb.

Women are more apt to alcoholic neuritis than are men. The quiet-steady drinkers are more liable to the disease than are open occassional drinkers. Delusion of suspicion is the most likely kind to be developed.

Anaesthesia is marked at the tips of the extremities more than up farther in the limb. Hyperesthesia to cold. Muscular sense usually well preserved.

Here muscular sense is affected.

The stomach must be stimulated to secrete up-some-thing that acts like alcohol: - red pepper or Tr. Capsicum.

Loss of memory.

2. Infectious cases: Diphtheria, typhoid, scarlet fever, measles, malaria, leprosy, beri-beri, grippe.

3. Rheumatic cases: Exposure to cold, overexertion.

4. Secondary to anæmia, tuberculosis, syphilis, septicæmia, general malnutrition, and diabetes.

Symptoms.—Acute or subacute onset with fever, 103°–104° F., and general febrile symptoms—sometimes without fever.

Sensory Symptoms.—First to occur. Pain—Sharp, severe, located in limbs, increased by motion or pressure; muscular and nerve tenderness, sensitiveness.

Paræsthesiæ—Burning, tingling, numbness, band about legs and body.

Anæsthesia—In tips of fingers and toes, extends up limbs.

Ataxia—In fine movements and loss of sense of position.

Motor Symptoms.—Paralysis. In extensors, dropped wrist and foot, general inability to walk or move in bed; contractures; cranial nerves may be paralyzed, causing symptoms in face.

Atrophy—Rapid in onset. R. D., or diminution in electric contractility; loss of deep reflexes; no paralysis of sphincters.

Trophic Symptoms.—Vasomotor paralysis; glossy skin; œdema; abnormal growth of nails and hair; sweating profuse; no bedsores; urticaria.

Certain cases present peculiar features in their course.

1. *Alcoholic Neuritis.*—(*a*) Rapid onset is the rule, with delirium and delusions, subsequently imperfect memory of recent events. Total paralysis, with very great pain; anæsthesia very marked, also tremor and tenderness; dropped wrists and feet; or (*b*) all symptoms of locomotor ataxia develop rapidly, except bladder and rectal loss of control. In both forms there is danger of heart failure or death from exhaustion, due to complicating gastritis. Usually there is rapid increase of symptoms for three or four weeks, and then, after a stationary period of two months, a slow progress to recovery, the duration being from six to twelve months.

2. *Lead Neuritis.*—Onset with intestinal colic, lead line on gums. Paralysis without sensory disturbance in hands, causing dropped wrists. May affect all muscles of arms and legs, and go on to sensory symptoms with delusions, but this is rare.

Gradual recovery, when poison is eliminated, in four months.

3. *Arsenical Cases.*—Gastric disturbance at outset. Then paralysis with ataxia, tremor, atrophy, and great numbness, but little

pain; legs and arms equally affected. Recovery in two to six months, depending on original severity.

4. *Coal-Gas Cases.*—Subacute and slight, chiefly sensory, numbness for a long time in fingers, hands, and feet.

5. *Diphtheritic Cases.*—Severe paralysis of all extremities, usually without sensory disturbance, cranial nerves frequently affected. It begins in uvula, and swallowing and talking become difficult. Eyes are often turned. Ataxia is marked in some cases. Never pain. Progress to recovery usual in three months.

6. *Epidemic Form.* (Beri-beri.) Occurs in Brazil, Red Sea, India, Borneo, Japan. Several types: acute pernicious, chronic œdematous, chronic atrophic. Too rare to need attention here. (See Gowers, I., 151; also New York *Medical Record*, February 12, 1887.)

Diagnosis.—From anterior poliomyelitis: by pain, tenderness along nerves, sensory symptoms, distribution of paralysis symmetrically. From locomotor ataxia: by rapid onset and paralysis, preservation of control of bladder, and absence of Argyle-Robertson pupil. From myelitis: by absence of affection of bladder and rectum, by absence of bedsores, and tenderness of spine to heat, and by distribution of symptoms in periphery.

Prognosis.—Good in large majority of cases; slow recovery; bad when heart becomes rapid and respiration poor.

Treatment.—Rest in bed. Warm applications by packs to limbs. Rubbing with oil; gentle massage if possible. Prevent deformities and contractures by proper position of limbs. Electricity, galvanism through nerves and to muscles after acute stage. Baths, warm, 98° F., for half-hour several times daily.

Attention to diet, nourish well, cut off alcohol in all cases.

General and nerve tonics; iron, quinine, strychnine.

In acute stage, sodium salicylate, gr. x., q. 3 hr., and sedatives, morphine, phenacetine.

In chronic stage, arsenic $\frac{1}{60}$ gr. t.i.d., cod-liver oil.

THE SPINAL CORD AND CRANIAL-NERVE NUCLEI.

THE spinal cord in man is made up of thirty-one segments—each of which consists of a mass of gray matter joined to a pair of spinal nerves. There is no apparent line of boundary between adjacent segments, the cord being a long cylindrical organ. But in snakes and rabbits the area of gray matter is much larger opposite the entrance of nerves than between them, and in fishes there is a visible boundary between adjacent segments.

Each segment presents a symmetrical arrangement of its two halves. Each half consists of a central collection of gray matter surrounded by white tracts. Each gray mass has an anterior horn, a central mass, and a posterior horn. To each horn nerve-fibres are joined making the anterior and posterior nerve-roots. The two halves are separated from one another in front by the anterior fissure, and behind by the posterior septum. They are joined together by the anterior white commissure and by the gray commissure in which lies the central canal of the cord. The functions of each segment are shown in the table on page 12.

THE ANTERIOR HORNS OF THE SPINAL CORD.

These vary greatly in size and shape in various segments, being large opposite the entrance of the cervical and lumbo-sacral nerves and thus causing the cervical and lumbo-sacral enlargements of the cord.

Their size and shape depend upon the number of groups of cells found at different levels; for the horns are made up of cells—large, polygonal cells with nucleus and nucleolus—collected into distinct groups.

Some groups of cells lie on the edge of the horn, making projections of the gray mass into the white columns. These groups are found in all the higher vertebrates and are not peculiar to man. Other groups lie within the horn and these are peculiar to man and

Segment.	Muscles.	Reflex.	Sensation.
2d and 3d Cervical.	Sterno-mastoid. Trapezius.	Hypochondrium (?)	Back of head to vertex.
	Scaleni and neck.	Sudden inspiration produced by sudden pressure beneath the lower border of ribs.	Neck.
	Diaphragm.		
4th Cervical.	Diaphragm.	Pupil 4–7 cervical.	Neck.
	Deltoid.	Dilatation of the pupil produced by irritation of neck.	Upper shoulder.
	Biceps. Coraco-brachialis.		Outer arm.
	Supinator Longus.		
	Rhomboid.		
	Supra- & infra-spinatus.		
5th Cervical.	Deltoid.	Scapular.	Back of shoulder and arm.
	Biceps Coraco-brachialis	5th Cervical to 1st Dorsal.	Outer side of arm and forearm.
	Brachialis anticus.	Irritation of skin over the scapula produces contraction of the scapular muscles.	Anterior upper two-thirds of arm.
	Supinator longus.		
	Supinator brevis.		
	Deep muscles of shoulder blade.	Supinator Longus.	
	Rhomboid. Teres minor	Tapping its tendon in wrist produces flexion of forearm.	
	Pectoralis (clav. part).		
	Serratus magnus.		
6th Cervical.	Brach. anticus.	Triceps.	Outer side of arm and forearm.
	Pectoralis (clav. part).	5th to 6th Cervical.	
	Serratus magnus.	Tapping elbow tendon produces extension of forearm.	Inside and front of forearm.
	Triceps.		
	Extensors of wrist and fingers.	Posterior wrist.	
	Pronators.	6th to 8th Cervical.	
		Tapping tendons causes extension of hand.	
7th Cervical.	Triceps (long head).		Inner and back of arm and forearm.
	Extensors of wrist and fingers.	Anterior wrist.	
	Pronators of wrist.	7th to 8th Cervical.	Radial distribution in the hand.
	Flexors of wrist.	Tapping anterior tendon causes flexion of wrist.	
	Subscapular.	Palmar. 7th Cerv.–1st Dorsal.	
	Pectoralis (costal part).	Stroking palm causes closure of fingers.	
	Latissimus dorsi.		
	Teres major.		
8th Cervical.	Flexors of wrist and fingers.		Forearm and hand; median and ulnar areas.
	Intrinsic muscles of hand.		
1st Dorsal.	Extensors of thumb.		Ulnar distribution to hand.
	Intrinsic hand muscles.		
	Thenar and hypothenar eminences.		
2d and 12th Dorsal.	Muscles of back and abdomen.	Epigastric. 4th–7th Dorsal.	Skin of chest and abdomen, in bands running around and downward corresponding to spinal nerves.
	Erectores spinæ.	Tickl'g mammary region causes retraction of epigastrium.	
		Abdominal. 7th–11th Dorsal.	
		Stroking side of abdomen causes retraction of belly.	Upper gluteal region.
1st Lumbar.	Ilio-psoas.	Cremasteric. 1st–3d Lumbar.	Skin over groin and front of scrotum, and narrow band down the front of the thigh and leg.
	Sartorius.	Stroking inner thigh causes retraction of scrotum.	
2d Lumbar.	Ilio-psoas. Sartorius.	Patella tendon.	Inner side of thigh.
	Flexors of knee (Remak)	Striking tendon causes extension of leg. 2d–4th Lumbar.	Outer side of thigh.
	Quadriceps femoris.		
3d Lumbar.	Quadriceps femoris.		
	Inner rotators of thigh.		Inner side of thigh and leg to ankle.
	Abductors of thigh.		
4th Lumbar.	Abductors of thigh.	Gluteal.	Inner side of foot.
	Adductors of thigh.	4th–5th Lumbar.	
	Flexors of knee (Ferrier)	Stroking buttock causes dimpling in fold of buttock.	
	Tibialis anticus.		
5th Lumbar.	Outward rotators of thigh.	Achilles tendon.	Lower gluteal region back of thigh. Leg and foot outer part.
	Flexors of knee (Ferrier)	Over-extension causes rapid flexion of ankle, called ankle clonus.	
	Flexors of ankle. Peronei		
	Extensors of toes.		
1st and 2d Sacral.	Flexors of ankle.	Plantar.	Leg and foot except inner side.
	Long flexor of toes.	Tickling sole of foot causes flexion of toes and retraction of leg.	
	Intrinsic muscles of foot		
3d to 5th Sacral.	Peroneus longus.	Bladder and rectal centre.	Perineum and back of scrotum. Anus.

the higher apes. Each group of cells presides over the action of a muscle and maintains its tone and nutrition, so that all motor impulses to the muscle, whether reflex, automatic, or voluntary, proceed from the group of cells. The outer groups govern muscles of flexion, extension, pronation, and supination of the larger joints. The inner groups govern the small muscles of the fingers and toes. Hence the outer groups are said to preside over fundamental motions and the inner groups over accessory motions.

The thumbless monkey has no inner groups in the cervical region. These are present in monkeys with thumbs and in man. When the inner groups are destroyed by disease, the finger motions are paralyzed.

In each segment there may be from two to eight groups of cells. Any group may extend through several segments. If a single segment is destroyed by disease several muscles will be paralyzed. But no muscle will be totally paralyzed unless its entire group of cells is destroyed. Total paralysis with complete atrophy in a muscle indicates destruction of the entire group of cells which govern the muscle. If the group extends through several segments the paralysis implies disease in all the segments. The table shows the situation in the various segments of the group of cells governing the various muscles, so far as these have been determined.

Anterior Poliomyelitis.

Anterior Poliomyelitis is a disease limited to the anterior horns of the spinal cord. It may be acute or chronic.

Acute anterior poliomyelitis is a common disease of childhood known as *infantile paralysis*. It occasionally occurs in adults. Chronic anterior poliomyelitis is an uncommon disease of adult life or old age, known as atrophic paralysis.

Acute Anterior Poliomyelitis. Etiology.—Occurs chiefly between two to five years, when exertion is greatest, in the summer, June to September, when exposure is greatest to heat, occasionally after falls and after fevers; it is probably an acute infectious disease in two-thirds of the cases. In the other third it is probably due to hemorrhage from or to thrombosis in a spinal blood-vessel.

Pathology.—Lesion is an acute inflammation of the entire gray matter of the anterior horn, the cells and interlacing fibres being affected. The cells become swollen, undergo degeneration, and

liquefy, and then shrivel and atrophy. The fibres are separated from their cells and atrophy. The neuroglia is infiltrated with inflammatory corpuscles. The capillaries are distended and frequently rupture. The process may go on to complete destruction of the cells or may be arrested at any stage.

If it is arrested early, repair may ensue and the cells regain their former appearance. If it goes on, the result is a diminution in the size of the horn, a change in its shape, and a contraction of the entire cord at the level of the lesion.

As a rule the entire anterior horn is at first involved, but the permanent destruction is limited to one or two groups of cells on one side only.

Inasmuch as each motor nerve-fibre comes from and is a part of the motor cell, the nerve-fibres degenerate when the motor-cells are destroyed. Atrophy of the motor nerve-roots and motor-nerves from the anterior horns to the muscles is therefore part of the lesion in anterior poliomyelitis. The deformity of the spinal cord at the level of the lesion and the atrophy of the nerve-trunks are due in part to these changes in the nerve-fibres.

The muscles which are paralyzed atrophy rapidly, and when recovery does not occur they remain small.

While no special groups of cells are known to control the nutrition of the bones, it is found that if the gray matter near the central region of the segment is destroyed by the disease, the bones are much retarded in their growth. Hence in a severe case of infantile palsy the limb affected is smaller than other limbs in all its parts. But trophic changes in the skin, such as sores, glossy skin, or gangrene, do not occur. Through the anterior horns pass fibres from the central region which control the vascular tone and metabolic processes. Hence in lesions of the anterior horn symptoms due to the destruction of these fibres ensue, viz., sluggish circulation, cyanosis, and lowered temperature, with imperfect nutrition.

Symptoms.—Sudden onset with fever 100° to 103° F., occasionally without. Febrile symptoms: Headache, loss of appetite, nausea, vomiting, diarrhœa. Restlessness: often convulsions and delirium. The fever rarely continues more than two to three days. Occasionally pain in the back and limbs is complained of when child is old enough to talk.

Paralysis appears suddenly. Sometimes is found in the morning; is complete and extensive either in one limb or more; legs

oftener than arms, 4 to 1; one limb more than the other. Maximum is reached in first week. Remains stationary for two to six weeks and then improves. Result is recovery, which is partial; some muscles remain paralyzed. The affected limb atrophies rapidly. Atrophy is most intense in the muscles permanently weak. There is a change in the electric reactions in the paralyzed muscle. R. D. Loss of faradic excitement in two weeks or sooner. Increase at first, then decrease, in galvanic excitability. Muscles contract slowly instead of quickly, more forcibly to the positive than to the negative pole.

The nutrition of the entire limb suffers; bones small and growth impaired; fat is less firm, and skin is cold; but no bedsores appear. The sphincters are not affected. The reflex action is lost at the level affected, not changed elsewhere.

Course.—There is a progressive improvement which begins within two months, but usually some muscles remain paralyzed and atrophied, and the joints moved by these may become deformed; spine curved. The muscles most frequently affected are: The peronei and tibialis anticus in the leg—hence talipes equinus and varus; the muscles of the calf and anterior part of the thigh; the deltoid, biceps, supinators, and spinati in the arm—hence subluxation of humerus; the muscles of the hand and forearm.

General health is unaffected.

In adults acute anterior poliomyelitis presents the same symptoms, but fever is less severe and pain is greater in muscles. Extent of initial paralysis is greater. Occasionally the onset is slow, so that case is called subacute, developing in two to four weeks progressively, remaining for a time stationary and then subsiding.

Diagnosis.—From acute rheumatism in infants. Rheumatism may produce immobility of joints. It also causes pain, redness, swelling of joints, and tenderness.

Prognosis.—Good as to life.

As to recovery, at *end of two weeks* test faradic reaction. The muscles in which it persists will recover entirely.

At *end of three months* test again. The muscles in which it has returned will recover partially.

At *end of six months* test again. The muscles which do not respond will never recover.

As to use of limb, it depends on growth of bone; *e.g.*, short leg on development of deformity and contractures; *e.g.*, talipes.

Treatment.—In acute stage: Cup or blister the spine and keep quiet. Give ergot and bromide, and antipyretics.

In chronic stage: Rubbing, warm clothing, galvanism to the muscles, interrupted, continuous. Strychnine $\frac{1}{100}$ to $\frac{1}{50}$ t.i.d., according to age. Prevent or remedy deformities by apparatus or surgical operation.

Chronic Anterior Poliomyelitis. — Progressive muscular atrophy occurs in adults between thirty and fifty years; is a rare disease.

Etiology.—Falls and injuries, exposure to damp and cold, alcoholic and sexual excesses, lead poisoning (?).

Symptoms.—Gradually increasing atrophic paralysis, several months in onset, preceded by fatigue and weakness and loss of weight. Muscles become flabby, then atrophied and paralyzed.

It begins in legs and extends upward (peroneal type).

It may begin in hands and then extend to shoulders and invade the back (Aran-Duchenne type).

Finally, all the muscles of the body, including face and tongue, may be affected. The tendon reflexes are lost.

There are fibrillary twitchings in muscles.

There are electrical changes which vary in different cases; usually R. D. is found.

The circulation is poor in the limb, skin cold and blue.

There is no sensory disturbance except slight numbness.

There are no visceral symptoms, no disturbance of bladder and rectal control. Bedsores do not develop.

Course.—Gradual increase after a long period of stationary state. Sometimes a steady progress to bulbar paralysis and death.

Treatment.—Similar to chronic stage of acute anterior poliomyelitis.

Cranial-Nerve Nuclei.

The groups of cells in the cerebral axis (medulla oblongata, pons Varolii, and crura cerebri) which give origin to the motor cranial nerves lie partly on the floor of the fourth ventricle (XII., VI., IV., III.), and partly deep beneath the floor in the formatio reticularis (XI., IX., VII., Vm.). These groups of cells are quite homologous to the groups in the anterior horns of the cord, and are subject to diseases whose pathology is the same of that of poliomyelitis.

Give fluid diet - mostly milk. Quiet position is very necessary. Dry cups are better than blisters for a child. Actual cautery is very good. Bromide is good - 5 grs. every 2 or 3 hrs. for child 2 or 3 yrs. old. Ergot is doubtful. Phosphorous + cod-liver oil - glycero-phos. of lime or soda - should be used after acute stage - when regeneration is in progress.

May be acute or chronic.
Acute are usually due to thrombosis or hemorrhage or Embolism in the pons or medulla.

The usual form extends from below upward.

In the majority of cases it is an external ophthalmoplegia. In externa alone it is a nuclear disease not of a nerve trunk.
The disease may be of apoplektic origin.

In general paresis & locomotor ataxia there is ophthalmoplegia interna.
No treatment for internal
1/60 - 1/30 gr. strychnine 3 x day for externa.

Bulbar Paralysis—or Glosso-Labio-Laryngeal Paralysis (*Rare*).

Symptoms.—Slowly progressing paralysis with atrophy in the motor cranial nerve nuclei muscles.

1. Disturbance of speech. Alalia, tongue letters, R L D T, due to paralysis of XII. nerve nucleus. Atrophy and tremor of tongue with paralysis. Chewing, swallowing impaired.

2. Muscles of lips and face become weak and thin, due to paralysis of VII. nerve nucleus. Alalia extends to lip letters, P B V F. Atrophy and tremor of lips with paralysis. Whistling, facial expression impaired. Saliva runs and is increased.

3. Muscles of pharynx and larynx affected, due to paralysis of the IX. and XI. nerve nuclei. Food regurgitates or cannot be swallowed; reflex action impaired; larynx unprotected; voice monotonous, low; cough impossible.

There is no change of sensation or of taste.

Pulse sometimes rapid, 100 to 130.

Duration, two to five years.

Death from inanition, pneumonia, heart failure.

Ophthalmoplegia.

This is a rare disease, due to an atrophy of the nuclei of the nerves moving the eyeball.

The muscular apparatus of the eye consists of external muscles moving the eyeball: Recti and obliqui and levator palpebræ. Internal muscles moving the iris: Dilators and constrictors. Each of these may be affected alone or together.

Ophthalmoplegia { externa. / interna.

Ophthalmoplegia Externa. Symptoms.—Gradually increasing loss of power in all the muscles of eyeball. One or more muscles at first, then others, till all are affected.

Result, ptosis. Various forms of strabismus, nystagmus, and finally immobility of eyeball. Double vision.

Ophthalmoplegia Interna. Symptoms. — Loss of reflex to light. Occurs as a symptom in locomotor ataxia and general paresis.

Diagnosis.—From paralysis of single nerves, III. nerve or VI. nerve, by the fact that such disease is unilateral and all the muscles

supplied by the single nerve are involved, others remaining unaffected, and by the fact that the pupil is always affected.

Progressive Muscular Dystrophy.

This is a primary muscular disease which results in atrophy and paralysis. It is commonly mistaken for chronic anterior poliomyelitis or progressive muscular atrophy, but differs from this in causation, symptoms, and pathology.

Causation.—It is an hereditary affection, or a disease of development or of nutrition. It develops in several children in same family with same or various types.

Pathology.—The lesion is an essential and primary change in the muscle fibres.

1. Individual fibres become hypertrophied.

The nuclei of the muscle fibres are increased in number.

Then vacuoles appear as evidence of degeneration and fibres split up.

At the same time there is an increase in connective tissue about the muscle fibres.

Sometimes there is a development of fat between the muscle fibres.

2. Subsequently to the hypertrophy and sometimes without it, atrophy of the muscle fibres occurs with same development of fat and connective tissue or without it.

Appearance during hypertrophy is a very fat limb in which muscles are really weak; during atrophy is a very thin limb in which muscles are very weak or even totally paralyzed.

Symptoms.—In all forms there develops a progressive simultaneous weakness and atrophy of the muscles affected, going on slowly to complete paralysis. The consequences of such paralysis are evident in size, posture, and deformities, use, motion, and gait.

No fibrillary twitching *vs.* progressive muscular atrophy.

No R. D. *vs.* chronic anterior poliomyelitis.

No sensory disturbance, bladder, or rectal, or trophic skin changes.

Various types of the disease classified by Erb:

I. Infantile muscular dystrophy.

1. Hypertrophic in type.

Pseudo-hypertrophy—fat only.

True hypertrophy, muscle also.

the theory:- the individual starts of muscle cells which will die before the individual ie the teeth - the ovary & the testicles.

these are the children of which mothers are usually proud & like to show their apparently strong & well developed limbs.

Pseudo hypertrophy:- most comon in children 2 & 8 yrs. old - more in males - usually more than 1 child in family is affected - usually hereditary. Begins in calf muscles - walk w. legs apart & just have difficulty in going upstairs. Muscles of leg then of back are affected. then shoulder-blade muscles. shoulders are thrown back.

2. Atrophic type.
   In limbs only.
   In limbs and face also.

II. Adult muscular dystrophy, with similar classes.

I. Infantile Muscular Dystrophy.—Hypertrophic Form.

1. *Pseudo-hypertrophic Paralysis.*—Children aged from two to eight affected, males more than females. Only twenty-five per cent. occur after twenty years. Several in one family.

Begins with difficulty in gait. Cannot walk, go up-stairs, or get up.

Muscles of back atrophied, and calves of legs hypertrophied. Also infraspinatus and deltoid hypertrophied.

Other muscles atrophy, pectorales, latissimus dorsi, triceps, and muscles of thighs.

Posture, shoulders far back. Gait is waddling, and mode of rising by climbing up the legs due to paralysis of extensors of hip is characteristic.

Finally, inability to sit up from paralysis of trunk.

After paralysis has occurred shortening of muscles follows, and deformities, talipes equinus, and curvature of spine.

No R. D. Knee-jerk normal, then weak. Sensation and sphincters not involved.

Death is due to some intercurrent malady, occurs between twelve and twenty years of age.

Only treatment is exercise and massage.

2. *True Hypertrophic Paralysis* (juvenile muscular atrophy). Develops in children six to ten. Several in a family. Begins with progressive weakness of muscles.

Muscles affected with hypertrophy are deltoid, infraspinatus, muscles of forearm, sartorius, gastrocnemii.

Muscles which atrophy are: pectorales, latissimus dorsi, serratus magnus, biceps, coracobrachialis, supinator longus, adductors of thighs, glutei and back muscles; finally, calf and peronei.

Gait, posture, and deformities are characteristic. No fibrillary contraction. No R. D. Knee-jerk slowly diminished. No disturbance of sensation or of sphincters.

3. *Atrophic Type in Body and Limbs* (hereditary muscular atrophy). Same features, except entire absence of hypertrophy. Develops from eight to ten years.

4. *Atrophic Type in Limbs and Face* (infantile muscular atrophy; Landouzy-Déjerine type). Face is often first affected.

When face is involved the zygomatic muscles are affected first; loss of naso-labial fold; flat mask-like face; lower lip projects; orbicularis is affected; lips open; labials can't be spoken. Sometimes orbic. palpebræ weak. Facial muscles atrophy slowly.

The other muscles of the body are affected in succession much later, pectorals, latissimus dorsi, biceps, and triceps. Deltoids may be hypertrophied. Back muscles and extensors of thighs are invaded.

Gait, posture, same as in other types.

II. Adult Muscular Dystrophy.

Any of these forms may occur in adults. Usually a family history can be obtained. Usually develops about age of eighteen, sometimes not till thirty. Some muscles are hypertrophied, others atrophied as in infantile types.

The Central Gray Matter of the Spinal Cord and its Diseases.

The portion of gray matter lying behind the anterior horn, and adjacent to the central canal of the spinal cord and extending backward to the posterior horn, is made up of gelatinous gray substance and of scattered nerve-cells with a plexus of fibres. It contains one long column of flask-shaped cells—the column of Clarke—in its posterior median part; and an irregular column of small cells in its lateral part—the intermedio-lateral column. The majority of these cells differ from the cells of the anterior horn, in not having a long axis-cylinder process which enters a nerve. They have numerous branching processes, some sending a long branch to the other side through the anterior commissure, others sending a branch out into the antero-lateral column where it bifurcates, turning up and down, and in its course gives off collaterals, which enter the gray matter at various levels. These cells thus form an associated system within the cord.

The function of the central gray matter is to preside over, 1, vasomotor tone; 2, nutrition of all parts; 3, visceral activity, including digestion and excretion, and the activities regulated by the sympathetic system.

1. Vasomotor innervation is a reflex matter requiring centres with sensory and motor-nerves. Gaskell has proven that these lie in the central gray matter and are joined by fine nerve-fibres which pass in the nerve-roots through the two horns of the cord. The regulation of the flow of blood in accordance with the need of an organ is governed here.

2. The proper degree of metabolism necessary for nutrition in the skin, bones, and joints and muscles, or for repair of tissue, is under the control of nerves whose centres are in the central gray matter.

3. The sympathetic system of nerves presides directly over digestion, circulation, secretion, and excretion, but is in turn controlled by spinal centres which lie in the entire length of the spinal cord in the central gray matter. See Gowers, I., pp. 205 to 209.

The central gray matter receives sensory impulses of pain and temperature sense, and transmits these for some distance on their way upward after they reach it through the posterior nerve-roots; each segment of the cord being related to its own part of the surface, as shown in the table.

All these functions are disturbed in diseases of the central gray matter.

Syringomyelitis.

Pathology.—This is a rare disease, usually of congenital origin, characterized by a development of gliomatous tissue in the gray matter around the central canal, which subsequently extends outward, involving the entire central gray matter, and then degenerates and is absorbed, leaving a cavity in the spinal cord. Such a cavity may extend through a part of the cord, or through its entire length; varying in size at different segments and thus destroying more or less of the gray matter. Its usual situation is in the lower cervical and upper dorsal regions. The disease may invade the anterior horns of the cord, causing the symptoms of chronic anterior poliomyelitis. It may invade the posterior horns and columns, causing symptoms of posterior sclerosis. Sometimes spinal gliosis develops in adult life and goes on to the formation of a cavity. Sometimes long perforating hemorrhages occur in the cord, and when the clot is absorbed a cavity remains. Sometimes local softening in the cord leads to the formation of a cavity. Any disease destroying the central gray matter of the cord causes the symptoms of syringomyelitis.

Symptoms.—1. Loss of the sensations of temperature and pain while the sensations of touch and location are retained. The distribution of this sensory disturbance depends on the location of the disease, *e.g.*, if the left hand and forearm only are thus affected, the disease must be limited to the left side of the cervical region of the cord in its lower third, which receives sensations from these parts. Such disturbance of sensation may be extensive, involving irregular areas of the body. It is never symmetrical on both sides.

2. Vasomotor and trophic symptoms. Cyanosis, œdema, blueness of extremities, with lowering of temperature, ulcers, bullous eruptions, sweating, thickened or glossy skin, defective growth of the nails, great brittleness of the bones leading to repeated fractures, and joint affections, also progressive atrophy of the muscles. These occur usually in one limb only.

3. Disturbance in and loss of control over the action of bladder and rectum, and loss of sexual power, are present when the lumbar region of the spinal cord is invaded.

4. Paralysis of the spinal muscles with curvatures of the spine.

Course of the disease is chronic, with slow progress till the symptoms are developed, often including the symptoms of chronic anterior poliomyelitis. It then remains stationary, but if it advances upward and invades the medulla, death may occur at any time from paralysis of the heart and respiration.

The prognosis is bad and no treatment can stop the course.

When tumors destroy the central gray matter or hemorrhages occur in it, the same terminal symptoms may be expected as in syringomyelitis.

The Posterior Horns of the Cord.

This portion of the cord is made up partly of gelatinous matter which occupies its extremity, and partly of a fine plexus of nerve-fibres, in which small cells are irregularly scattered. The function of the posterior horn is to receive the impulses coming in through the posterior nerve-roots, and to transmit such impulses in various directions. These are the sensations of touch, temperature, pain, pressure, and location, and of the muscular sense.

Each nerve of sensation gives off numerous branches on entering the cord, and finally terminates in a brush-like expansion of nerve-fibrils which surrounds some nerve-cell, or in the plexus of nerve-fibrils which make up a fine felt-like tissue in the posterior horn.

3. They make up the pyramidal + cerebellar tracts &
Gowers Column, etc.

The columns of the cord are not exclusively sensory or
motor - they are preponderately so but contain also
other fibres.

Any fibre after entering the posterior nerve-root may pass directly into the posterior horn, or may turn up or down in the white matter near the horn and enter the posterior horn at some level other than that of its entrance. The posterior horns are continuous upward (through the medulla and pons) with the ascending root of the fifth nerve. The diseases of the posterior horn will be considered with diseases of the posterior roots and columns, as they uniformly coincide.

The White Columns of the Spinal Cord and their Diseases.

The gray matter of the spinal cord is surrounded by white tissue made up of anterior and posterior nerve-roots and of nerve-fibres passing up or down the cord for longer or shorter distances. The white columns contain therefore:

1. Fibres joining the skin, joints, muscles, and viscera to the gray matter of the spinal cord and entering the various segments through the anterior and posterior nerve-roots.
2. Fibres connecting the various segments with one another.
3. Fibres joining the various segments with the brain.

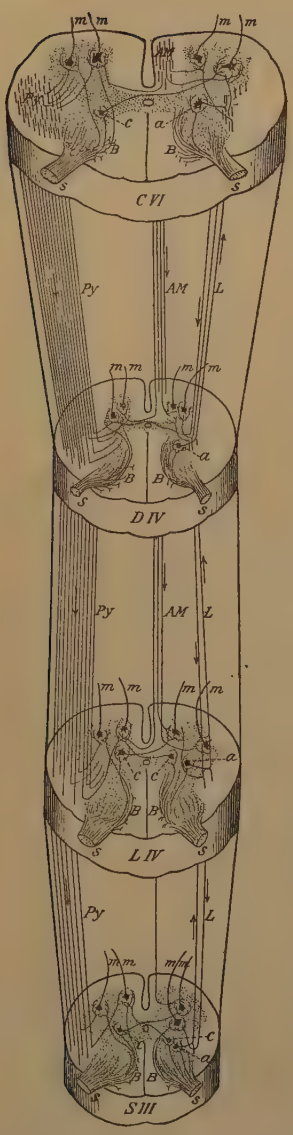
The white matter can be divided into different columns by observing the process of development—different columns develop at different periods of fœtal life; or by studying the process of disease —different diseases select different columns.

These columns and their diseases are as follows:

The Motor Columns.

1. *Anterior median column.*
2. *Lateral pyramidal column.*

At the lower limit of the medulla each anterior pyramid divides into two parts; one part passes directly on into the anterior median column of the cord; the other part crosses over to the opposite lateral column of the cord, lying near to the gray matter in the angle between the anterior and posterior horn. As the fibres making up these columns pass down the cord, they turn into the anterior gray horns of the different segments—each column becoming smaller the lower it goes. The anterior median column is exhausted at the lower dorsal cord, and therefore does not exist in the lumbar region. The lateral pyramidal column extends to the lowest segment



of the cord. These columns transmit motor impulses from the motor cortex of the brain and from the motor ganglia of the brain to the spinal motor centres. These columns also contain fibres whose origin is in the pons and cerebellum, which pass with the motor fibres from the brain and terminate in the anterior horns of the cord.

They also contain fibres of association passing between various segments of the cord. All these sets of fibres degenerate downward. The area of degeneration is less in extent when the lesion is in the brain than when it is in the cord itself.

Lateral Sclerosis.

Lateral sclerosis is a chronic sclerotic process affecting the lateral pyramidal columns of the spinal cord.

It may be *primary* and is then bilateral.

It may be *secondary* to other diseases in the brain or cord which separate the fibres of the motor tract from their centres of origin and nutrition in the brain cortex. Such diseases are:

1. Tumors, softening or hemorrhage in the brain, destroying the motor tract in one hemisphere, in which case the secondary sclerosis is found in the anterior median column and the opposite lateral pyramidal tract.

2. Congenital maldevelopment of

Fig. 1.—The Motor Tracts. *Py*, lateral pyramidal; *AM*, anterior median column, both from right pyramid of medulla to motor neurons of anterior horn, whence motor nerves (*m*) pass to muscles; *s*, sensory nerve entering posterior horn and posterior root zone of the column of Burdach (B), and sending fibres to (*a*) association-neurons; (*c*) commissural neurons, and also to motor neurons, in anterior horn; *L*, fibres of lateral tract consisting of association-fibres between various segments.

Retention of urine is due to a weak detrusor muscle in combination w/- a strong sphincter.

the brain and hydrocephalus, affecting both motor tracts, in which case both anterior median and both lateral columns are sclerotic.

3. Any lesion in the spinal cord, either unilateral or bilateral, or compression of the cord by disease of the vertebræ, in which case the sclerosis only appears below the level of the lesion, and its extent is greater in area than in 1 or 2.

Pathology of Sclerosis.—1. An increase in the thickness of the connective-tissue framework which supports the nerve-fibres. 2. A primary degeneration or a secondary destruction of the axis cylinder in each nerve-fibre, resulting in its swelling, segmentation, fatty degeneration, and final absorption. The result is to reduce the number of fibres in any column of the cord, or even to destroy them entirely, the place of the fibres being filled by connective tissue.

Symptoms of Primary Lateral Sclerosis, or Spastic Paraplegia.—A weakness and stiffness in motion, beginning in the legs and ascending, finally involving the arms. The muscles affected do not atrophy rapidly, as their spinal centres are not diseased. Their reflex and mechanical excitability is increased, as the spinal centres are no longer inhibited by brain impulses. Hence increased knee-jerk and ankle clonus are present. They become rigid from the increased spinal activity. Hence the gait is at first "spastic" (toes drag, knees overlap, motion stiff); finally, when paralysis is complete, the legs are drawn up, knees overlapping and so rigid as to be incapable of passive motion. There are no electric changes in the muscles.

The action of the bladder is imperfectly controlled, and finally involuntary emptying of the bladder at intervals or retention of urine occurs. Constipation is obstinate. In some cases of primary lateral sclerosis the bladder and rectum are not affected. The vascular tone is decreased toward the close of the disease and nutrition is impaired. There are no sensory symptoms.

Course of the Disease.—The symptoms develop slowly and increase until, after many years, permanent paralysis has developed.

Secondary Lateral Sclerosis presents the same symptoms on one or both sides, but the symptoms develop more rapidly according to the nature of the primary disease.

Diagnosis between lateral sclerosis and anterior poliomyelitis:

1. Lesion in pyramidal tracts.	Lesion in anterior gray horns.
2. *Paralysis* usually on both sides equally, in legs, or in legs and arms, never in arms alone.	*Paralysis* may be limited to any single limb, and rarely affects both limbs equally.

3. All muscles are about equally affected. No muscles are entirely normal.	Certain groups of muscles only are affected. Others escape wholly.
4. Muscular tone is heightened.	Muscular tone is diminished.
5. Tendency to rigidity appears.	Muscles are relaxed.
6. Reflex excitability is increased.	Reflex excitability is lost.
7. *Atrophy* is absent, or is slight; and merely due to disuse, hence is gradual in progress. It affects the entire limb.	*Atrophy* is always present in the paralyzed muscles. It advances rapidly, and may become extreme.
8. *Electric contractility* is unchanged.	*Electric contractility* is changed. Reaction of degeneration is present within two weeks of the onset.
9. *Vascular tone* is diminished; cyanosis and œdema may occur.	*Vascular tone* is diminished, but œdema does not occur.
10. Paralyzed limb is cold, and sweat may be increased.	Paralyzed limb is cool, but sweat is not increased.
11. *Trophic disturbances* in the skin are not infrequent.	*Trophic disturbances* in the skin do not occur.
12. The control over the bladder and rectum may be diminished or lost.	The control over the bladder and rectum is not impaired.

Treatment.—None. [Read Strümpell on spastic spinal paralysis and on compression myelitis.]

Amyotrophic Lateral Sclerosis.

This is a chronic disease affecting the entire motor system of the spinal cord and cerebral axis, both the groups of cells in the anterior horns and medulla and pons, and also the lateral pyramidal columns in the cord and in the medulla and pons being affected together. The extent of the sclerosis is greater in the cord than in primary lateral sclerosis, as the white column which lies between the pyramidal tract and the gray matter (the *lateral limiting column*) is also sclerotic in this disease. The changes in the anterior horns are the same as in chronic anterior poliomyelitis.

Symptoms.—The symptoms present features similar to those of chronic anterior poliomyelitis and of extensive secondary lateral sclerosis. Weakness, paresis, and atrophy begin in one or both arms and hands and increase slowly. The muscles of the hands and the extensors of the wrist, the deltoid and triceps, are the muscles chiefly affected. The electric contractility is diminished, and finally reaction of degeneration appears. The muscular irritability and reflexes are much increased. Finally contractures result.

Gradual onset of spastic paraplegia in the legs occurs a few

There are fibrillary twitchings in the muscles affected.

Irregular Forms:-

If symptoms begin in the legs and extend to the arms there is little spastic rigidity in the arms.

When the disease begins in the medulla - it simulates bulbar paralysis but af- it are increased reflexes of arms and legs in the earlier stages.

Differentiate from:

Tachymeningitis Cervicales Hypertrophica:

Pain present - radiating into arms hands & fingers.

" difficulty in motions of the head - so that head is thrown forward & held rigid.

months after the arms are affected. Spastic gait develops with increased reflexes. In the final stage the legs are totally paralyzed. There are no sensory disturbances, and bladder and rectum are usually under good control.

After several years symptoms of bulbar paralysis gradually develop, with paresis and atrophy of face, tongue, and throat, and death occurs from respiratory paralysis. Occasionally the bulbar symptoms appear early.

The course is a slow one. The disease may be arrested, but recovery is impossible.

Diagnosis from myelitis of the cervical region and from syringomyelitis by the absence of sensory symptoms; from anterior poliomyelitis chronica by increased mechanical and reflex excitability in muscles atrophied, and by development of spastic gait.

Treatment.—None. Strychnine gives relief but course is not stopped. Use of massage & electricity.

The Sensory Columns.

3. *Posterior Lateral Column, or Column of Burdach.*
4. *Posterior Median Column, or Column of Goll.*
5. *Marginal Column, or Column of Lissauer.*

These columns are made up chiefly of sensory fibres which enter the cord in the posterior nerve-roots. They also contain fibres of association between different segments of the cord. Their subdivisions will be better understood by a study of the course of the posterior nerve-roots.

The posterior nerve-roots contain two sets of fibres. The majority arise from the cells of the posterior spinal ganglia. The minority arise from the so-called tactile corpuscles of the extremities. Each spinal ganglion cell has two branches, or two divisions of one branch; one going out to the surface, the sensory nerve; the other going in to the cord, the posterior nerve-root. The cell in the ganglion nourishes both branches, and if either branch is separated from its cell it degenerates in its entire length. The sensory cranial nerves (X., IX., V.) arise from similar ganglia (jugulare Gasserian).

The posterior nerve-root enters the cord opposite to the apex of the posterior horn and then divides into two bundles, a median bundle and a lateral bundle.

(1) The median bundle containing large fibres enters the column of Burdach, forming its root-zone or *lateral zone.* Here a few fibres

turn downward into the segment next below, and end in the posterior horn; but the large majority of fibres turn upward to go to higher segments. Some of these soon enter the inner side of the posterior horn (short fibres). These lie near the horn in the lateral zone. Others pass up through several segments (medium fibres). These lie nearer to the median line and in the upper cord are adjacent to the column of Goll, forming the *middle zone* of the column of Burdach. The remainder pass all the way up to the medulla (long fibres), lying at first in the *hinder zone* of the column of Burdach and later forming the column of Goll. For as each root, from below upward, enters the cord, it displaces inward and backward the fibres already ascending; so that the higher the level the larger the number of ascending fibres. In a cross-section of the cord in the upper cervical region the long fibres which have come from the lumbo-sacral region occupy the column of Goll, the sacral fibres lying behind the lumbar; the long fibres from the dorsal region occupy the hinder zone of the column of Burdach; the long fibres from the cervical region occupy the middle zone of the column of Burdach. It is evident that there is no physiological distinction between the columns of Goll and Burdach. In the anterior part of both columns near the gray commissure lie association fibres and commissural fibres. It is thus evident that the median bundle

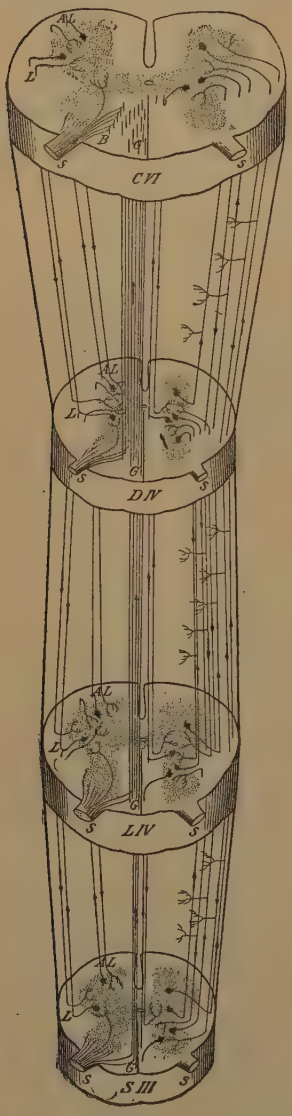


FIG. 2.—The Columns of Burdach and Goll are shown on the left side of the cord. The association-fibres of the cord joining the various segments with each other of various lengths are shown passing through all the columns. The oval column of Flechsig is shown in the lumbar and sacral regions, also called the descending septo-marginal tract.

of posterior nerve-root fibres constitutes the columns of Burdach and of Goll.

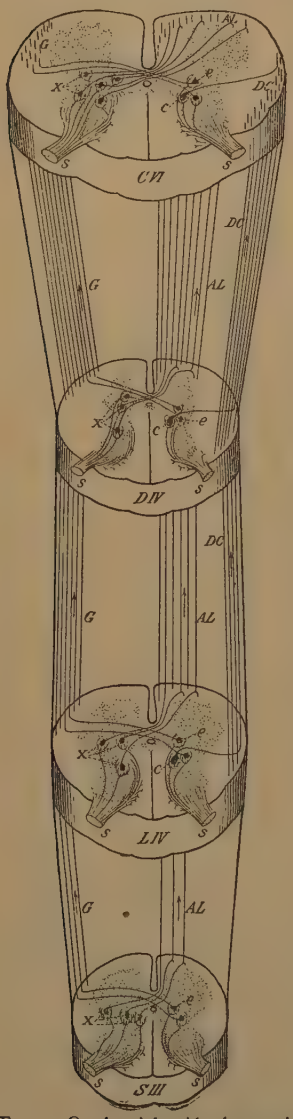
The final termination of these fibres is as follows: Each fibre on entering the cord bifurcates, a short branch turns down, a long branch turns up. The long branch in its course upward gives off collateral branches which pass into the gray matter of the posterior horn, and finally the branch itself ends there. In all cases the termination is by means of a brush-like expansion of nerve-fibrils. The short fibres end in the fine network making up the substantia gelatinosa of Rolando in the posterior horn, and about the cells of the posterior and central gray matter. The medium fibres end in a network which surrounds the cells of the column of Clarke in the central gray matter. A few fibres pass forward to the anterior horn and to the opposite side through the posterior gray commissure. The long fibres pass up to the nuclei gracilis and cuneatus in the medulla.

(2) The lateral bundle of the posterior nerve-root consists of both large and small nerve-fibres. Many of these pass directly into the gray matter of the posterior horn, where some end in a network of fibres about the cells of the gelatinous substance, or deeper in the horn; and others pass forward and cross over in the gray commissure to the opposite side, where they turn outward into the antero-lateral column or backward into the column of Goll. The smaller fibres bifurcate on entering the cord and turn down and up, forming the column of Lissauer, in which they pass but a short distance and then turn into the horn. This column is of uniform size through the cord, and hence cannot contain any long fibres.

6. *Antero-lateral Ascending Tract or Column of Gowers.*—In the antero-lateral column of the cord lying on the periphery just in front of the direct cerebellar column is a small tract which ascends through the cord and enters the formatio reticularis of the medulla, where it appears to end in the gray cells of this formation. It begins in the lumbar region, and increases in size as it ascends, receiving fibres from the cells of the central gray matter. It degenerates upward. It has been supposed to convey sensations of temperature and pain upward after they have passed through the central gray matter.

7. *Direct Cerebellar Column.*—This column lies on the lateral periphery of the cord just outside of the pyramidal tract and be-

hind the antero-lateral ascending tract. It is very small in the upper lumbar region, becomes larger in the dorsal region, and is of considerable size in the cervical region. Its fibres come from the cells of the column of Clarke in the central gray matter. It passes into the restiform body in the medulla and through this into the cerebellum, where it ends in the vermiform or middle lobe. It is supposed to convey sensations of equilibrium in the act of walking.



It is evident that the sensory fibres from the posterior nerve-roots are distributed to many different columns of the spinal cord, and even in the medulla have numerous different connections. This anatomical fact may be associated with the physiological fact that one sensation is capable of producing several different effects, *e.g.*, a burn causes a reflex withdrawal of the part burned, vascular changes, and a process of repair, sensations of heat, touch, and pain, accurately localized, followed by conscious effort to protect the part: *e.g.*, stumbling in the act of walking causes reflex balancing movements involving motions of the entire trunk and arms to reëstablish the centre of gravity or protect against fall, also conscious sensation of unstable position, possibly vertigo, quickened respiration, action of ocular muscles and head to observe danger, sensation of contact, possibly pain, in the toes, and corresponding vascular changes locally, and finally

FIG. 3.—On the right side the termination of Sensory Nerves about the Cells of Clarke's Column (*C*) and about the Intrinsic Cells (*e*) is shown. From C fibres pass up in *D C*, the direct cerebellar column. From *e* fibres pass over to the column of Gowers (*G*). On the left side the termination of sensory fibres about intrinsic cells (*x*) is shown. From *x* fibres cross to the antero-lateral column and ascend.

Strau thinks that there is a primary degeneration of the spinal ganglia cells + a secondary degeneration of the columns of the cord involved.

voluntary effort to correct position after an appreciation of all the various sensations received.

We distinguish conscious sensations of contact, temperature, pain, muscular sense, including pressure and position, and unconscious sensations, which set up reflex and automatic acts, vascular changes, variation in thermic and nutritive processes, and changes in muscular tone. These all enter by the posterior nerve-roots, and set up activities in the central nervous system.

Locomotor Ataxia.

This is a chronic disease of the sensory part of the nervous system, the posterior nerve-roots and the posterior columns of the cord being diseased. It is also called *tabes dorsalis*, or *posterior spinal sclerosis*.

Pathology.—There is a degenerative neuritis of the posterior nerve-roots, and sometimes of the sensory part of the peripheral nerves. There is a sclerosis of the posterior columns of the spinal cord, beginning in the lateral or root-zone of the column of Burdach and in the column of Lissauer, extending thence to the middle zone of the column of Burdach, then to the column of Goll, and finally to the hinder zone of the column of Burdach. Thus both posterior columns are eventually diseased in their entire extent. The disease usually begins in the lumbo-sacral region and extends upward through the cord. It is many years before it invades the cervical region or destroys the entire area of the posterior columns. The terminal network of nerve-fibres in the posterior gray matter, and especially about the column of Clarke, is destroyed by the disease.

Theories of the disease: (1) It is a primary sclerosis of the posterior columns of the cord. (2) It is a sclerosis of the root-zone, due to disease of the vessels entering this zone. (3) It is a primary disease of the cells of the posterior spinal ganglia, with secondary degeneration of the sensory fibres entering the cord from the ganglia, and compensatory sclerosis in their course.

Etiology.—It is a disease of adult life, persons under twenty-five rarely being affected, and it is rare among women. The chief predisposing cause is syphilis, which precedes it in seventy per cent. of the cases. The exciting causes are great physical exertion, exposure to cold and wet, and alcoholic and sexual excesses.

Symptoms.—The symptoms of the disease are very numerous, but appear in succession. They may be classified according to the time of their appearance. There is a stage of pain, a stage of ataxia, and a stage of paralysis.

Symptoms in Stage of Pain.

Paræsthesiæ. Numbness, formication, sensation of dead extremities, cotton or pins in soles of feet and fingers, coldness, itching of anus and scrotum or of other parts.

Pain. Short, sharp, cutting, boring. Deep pain; comes in attacks, one second to one-half minute repeated, or in severe attacks for several hours or days with intermissions, rarely continuous attacks. Begins in thighs and legs. Pain is a prominent symptom early, but may continue through all stages. It may be mistaken for rheumatism or sciatica, but its character is different. It is the same as in alcoholic multiple neuritis, but there tenderness exists. There is no relation between intensity of pain and degree of ataxia. As disease advances upward the pains extend. Pain may be absent or insignificant in some cases, especially in cases beginning with optic-nerve atrophy.

Pain in the small of the back and loins of an aching character may occur.

Girdle sensation or sensation of tightness and pressure intense and severe, about the legs and body. This ascends gradually as the disease advances.

Loss of knee-jerk, called Westphal's symptom.

Imperfect control of bladder. Slow urination. Slight dribbling, and hasty urination. Finally control is very imperfect and may be painful. This may be a dangerous complication, since death from cystitis occurs.

Impotence; occasionally priapism and sexual excitement precede this.

Constipation; usually obstinate throughout.

Loss of pupil reflex to light, the reaction in accommodation being preserved—Argyle-Robertson pupil.

Contracted pupil, "myosis spinalis," is frequent but not constant.

Symptoms in Stage of Ataxia.

In addition to those of first stage there develop:

Anæsthesia and analgesia. Sense of pain impaired and delayed. This may be tested with faradic brush. It prevents injuries from being perceived; hence ulcer of foot and Charcot joints occur as

The paraesthesia is referred to the surface of the body but the symptom is due to an irritation of the nerve at its entrance to the spinal cord. Pains are referred in the same way to the periphery.
The pain is usually first referred to the anterior surface of the thigh.

In the girdle there is usually some change in sensation.

Patients are usually more sensitive to cold & less sensitive to heat.
Guard against hot applications to & pressure the limbs.
Anaesthesia usually first appears on the trunk. It is present less often than usually stated. It is not uncommon in soles & feet, around anus & scrotum.

complications. Hence, guard against accidents in giving hot baths, blisters, etc.

Hyperalgesia. Sensation of pain may be produced by any touch, and pain is felt severely. This occurs in a few cases early in the disease.

Change in temperature sensation. Cold felt keenly and heat less than normal.

Complete anæsthesia develops late in the disease in the extremities.

Impairment of muscular sense. Muscular sense is the sense by which the situation and movements of the limbs are appreciated, and pressure and weight are felt. Not same as effort sense. Imperfect perception of weight and pressure is a symptom observed late. Situation of limbs is imperfectly perceived. Hence, one means of perfect guidance of limbs is removed. Hence, patient walks badly in the dark, and cannot touch objects accurately with eyes closed; hence he watches his own motions—aiding his muscular sense by sight. Therefore a blind ataxia is the most helpless of all.

Ataxia is imperfect co-ordination of muscular action. For every compound act a regular succession and proper degree of motions are needed, *e.g.*, closure of fist, walking. This regulation is largely automatic and is carried on in subcortical centres, which receive unconscious sensations through the posterior nerve-roots. It may be voluntary, by an effort of attention, and is so at first, for all motion, and always for highly complex motion, *e.g.*, writing. Hence ataxia of movement may occur in many diseases. It is a prominent symptom in locomotor ataxia.

Romberg's symptom—swaying when standing with eyes closed.

Ataxic gait—legs wide apart, feet lifted too high, and planted too forcibly, steps being of irregular length and body imperfectly balanced.

Imperfect use of hands in dressing, writing, etc. Irregular contraction of muscles on effort. Imperfect muscular tone.

Tests of ataxia. Stand with eyes closed, walk, turn suddenly or walk backward; touch heel to toe, or heel to knee; touch finger to nose or both fingers with eyes closed; pick up pin; button clothes.

Symptoms occasionally present, due to complication of neuritis in various nerves. Optic-nerve atrophy, with progressive blindness, develops in twenty per cent. of the cases. Optic disc looks white,

then gray. Arteries appear small and walls thin. Visual field is contracted for colors and for light. Finally total blindness occurs.

Paralysis of the ocular muscles.

VI. n. paralysis; convergent strabismus, with contracted pupil.

III. n. paralysis; divergent strabismus, with dilated pupil and ptosis. Nystagmus rarely occurs.

Deafness develops in a few cases from atrophy of auditory nerve.

Crises—(1) gastric; sudden vomiting with pain, for several hours, even for days. Great prostration follows. Rectal feeding necessary. Intestinal and rectal crises with diarrhœa and tenesmus have been observed. (2) Laryngeal; sudden and severe cough, with spasm of larynx and suffocation. (3) Cardiac; attacks of angina pectoris. All due to a complicating peripheral neuritis in pneumogastric nerve. These symptoms are quite exceptional.

Trophic disturbances are more common in stage of ataxia.

Charcot joints. Knee, elbow, ankle, wrist. Enormous swelling with effusion without pain. Erosion of ends of bones and destruction of articulation. Due to injury.

Perforating ulcer of foot, caused by a corn, or a subcutaneous hemorrhage, or a caries of bone neglected.

Rarefication of the bones with production of fractures.

Irregular muscular atrophies develop late in the disease.

Herpetic eruptions and pemphigus.

These are due to a combination of causes, chiefly to a loss of pain sense which prevents injuries from being perceived, or to a complicating peripheral neuritis.

Symptoms in Stage of Paralysis.

General nutrition remains good, but ataxia becomes so extreme as to render patient helpless and confine him to bed.

Control of bladder completely lost, and urine dribbles constantly.

Accidents produce injuries, and trophic disturbances follow.

Hence patients die of bedsores, cystitis, exhaustion, or of complicating pneumonia.

Course of the disease is chronic through the three stages, lasting twenty to forty years.

Usual mode of onset is with pain, loss of knee-jerk, bladder trouble, impotence, and ataxia.

Occasional modes of onset: (1) blindness, loss of knee-jerk, imperfect gait, numbness, but no pain.

Course of Loc. Ataxia: -

Some are acute cases - 3 to 6 mos. in onset
Others run course in 1 or 2 yrs. - ending in death.

Diag:- Phys. Signs are loss of knee-jerk - Argyle-Robertson Pupil - Romberg Symptom.

Fränckel Method for ataxia:- trace lines along floor w- toes forward & backward - then outward - inward & forward triangular movement - then a semicircle. Do this 4 times day - each motion 10 times & 10 minutes rest between each.

Antisyphilitic Treat. food where pts. have not had the treatment before. Don't give it to any pt. unless his physical condition is particularly good.

Saucheim - La Malou - Baden Baden.

Glycerophos. Lime Soda - for nerve energy & nutrition.

Deformities of the feet develop - not a true club foot - but similar to it. The deformity may prevent the pt. from standing.

Mental symptoms may develop.

Strabismus may be present.

(2) Gastric crises at intervals, loss of knee-jerk, then pain and ataxia.

(3) Various forms of strabismus, myosis, then ataxia of arms.

There are remissions in the symptoms and complications may subside, but no true arrest occurs or recovery, after the stage of ataxia has begun.

Diagnosis. Early, by means of pains, fatigue, loss of knee-jerk, and Argyle-Robertson pupil. Late, by ataxia and bladder disturbance.

Diagnosis from multiple neuritis. See page 10.

Treatment. Moderate exercise, avoiding all fatigue. Good general diet without special restrictions, which weaken patient. Spinal douches, tepid or cool, never extreme. Massage. Practise in fine movements to overcome ataxia. Medicines—arsenic, nitrate of silver, ergot.

Treatment of pain by antipyrine, etc., gelsemium, opium, faradization, hot applications. Of crises by counter-irritation and morphine. Of optic atrophy by strychnine. Of trophic disturbances by rest and apparatus.

Combined Sclerosis.

This is a Sclerosis of the Lateral and Posterior Columns (*rare*).

(1) Congenital cases. *Friedreich's Hereditary Ataxia* is due to defective development of the lateral and posterior columns. It occurs in several members of a family, and is hereditary.

Symptoms.—As the children grow up they are found to be awkward. They walk badly, and do not use arms well. Reflexes are lost, exceptionally exaggerated. Dull pains in legs, not lightning in character. No bladder symptoms, and no trophic changes, and no crises. Ataxia is marked and goes on to actual paralysis. Speech may be thick and nystagmus present. The condition is a chronic one lasting during life.

(2) Acquired cases. *Ataxic Paraplegia* of Gowers.

Etiology.—Occurs in males chiefly, between thirty and forty years of life. Not after syphilis, but after exposure to cold or sexual excess. Possibly after concussion of spine.

Symptoms.—Slow onset of weakness and ataxia together. Undue fatigue, unsteady gait. Romberg symptom. Dull pain in back and legs, no lightning pains. Reflexes increased. Knee-jerk high,

ankle clonus present. Sexual power lost. Sphincters less impaired than in tabes. Eye symptoms very rare. Tremor of face and speech sometimes develops.

Differs from tabes in fact that the nerve-roots are not involved.

Course.—Weakness increases slowly to paralysis. Rigidity soon develops, and contractures. Death occurs from complications.

No treatment.

The Association Columns.

8. *Antero-Lateral Column.*—There is a large collection of nerve-fibres forming a white column lying adjacent to the gray matter of the cord, in front and inside of the pyramidal tract, and inside of the direct cerebellar and antero-lateral ascending tracts, extending as far forward as the anterior nerve-roots. (See Fig. 1.) A portion lying next to the gray matter has been named *the lateral limiting layer.* This column contains a few fibres which degenerate all the way upward to the medulla, and it is possible that some sensory impulses are transmitted through it. (See Fig. 3.) The major part of the fibres are, however, short, and serve to connect the various segments of the cord with one another. (See Fig. 2.) In it pass the majority of the "association fibres" of the cord which harmonize the action of different parts.

9. *Anterior Column.*—This lies between the exit of the anterior nerve-roots and the anterior median column. It has the same function as the antero-lateral column, and like it ends in the formatio reticularis of the medulla.

There are very many polygonal cells scattered through the gray matter in both horns and in the central gray which send out to these columns an axis-cylinder process, which on entering the column bifurcates, sending one branch down and the other upward. These branches give off collaterals in their course, which pass into the gray matter at various levels and terminate in brush-like expansions. The original branches terminate in the same way after having a longer or shorter course. Thus the majority of the fibres in these columns are true association fibres. There are a few such fibres also in the portion of the posterior columns adjacent to the gray commissure.

Senile Paraplegia:-

Occurs chiefly in old people.
Gradual onset of weakness & more or less ataxia - etc. - symptoms are almost identical w/ - Ataxia Paraplegia.
No eye symps - no tremor of face or speech

Senile Paraplegia -

Resembles ataxia Paraplegia - symptoms are the same. Gradual onset of weakness & ataxia. No eye symptoms. No tremor of face or speech. Symptoms confined to the legs & dont extend to arms - more comm. in women - begins at menopause or later in life. in men after 50 yrs. Onset slow - takes 3 to 4 mths. to develop. Sometimes occurs in 2 weeks.
Starr's Theory:- malnutrition of cord due to venous congestion in lower part - usually have varicose veins of leg.
Put pt. to bed - massage - elevate legs - tonics & stimulants & it can be cured.
Girdle sensation is often present. This condition may go to develop a syringomyelitis.

May affect only a segment.
" " an entire enlargement.
" be scattered through cord, at
different levels.

Secondary Degenerations.

When the cord is divided by an injury or by transverse myelitis there results a degeneration in the various columns.

The anterior median and lateral pyramidal columns degenerate downward for their entire length. There is also a short tract of degeneration downward in the lateral zone of the column of Burdach. The anterior and antero-lateral columns degenerate downward for a short distance.

The columns of Burdach and Goll and Gowers and the direct cerebellar column degenerate upward through their entire length. The columns of Lissauer and the anterior and antero-lateral columns degenerate upward for a short distance.

Such degenerations are due to the fact that the transverse lesion cuts off the individual fibres from their nutrient cells, and hence the distal end degenerates, the fibres nourished by cells above the lesion degenerating downward and those by cells below the lesion degenerating upward.

Myelitis.

This is an inflammation of the spinal cord involving an entire segment, both gray and white matter.

Pathology.—Cord looks normal, but feels soft at points; or may break in two and be soft enough to be fluid. On cutting it the cut surface swells out as if semifluid; the gray matter looks red and capillary hemorrhages are seen; the limit of white and gray is indistinct.

Microscopically. — Large numbers of inflammatory corpuscles everywhere. Nerve-cells and fibres swollen and degenerated, granular and disappearing or atrophied. Neuroglia cells increased. Deiter's spider cells present. Fatty and granular cells everywhere as evidence of degeneration. Vessels are distended with blood and ruptured; walls may be infiltrated and thickened.

Secondary degeneration as already enumerated.

Etiology.—Males more than females. Soldiers, porters. Age, ten to forty. Exposure to cold. Overexertion. Falls and blows. Concussion of cord. Sexual excesses. Menstrual suppression. After confinement. Injuries of the cord. Compression of the cord

by disease of spinal bones or tumors. Secondary to acute infectious diseases. Syphilis and chronic alcoholism.

Varieties, General.—Entire cord affected—ascending or descending.

Disseminated.—Various segments at different levels affected.

Transverse.—One or two segments at one level affected.

Symptoms.—(1) Direct, due to destruction of cord tissue.

1. Motor. Paralysis, atrophy. R. D. Loss of tone in muscles.

2. Reflex. Loss of spinal reflexes. Paralysis of the bladder and rectum, impotence.

3. Sensory. Numbness. Pain. Paræsthesia, anæsthesia. Hyperæsthesia at upper level of lesion. Pain and tenderness of back to heat.

4. Vasomotor and trophic. Imperfect circulation. Skin cold. Sweat profuse. Bedsores on buttocks, sacrum, and heels.

The distribution of the direct symptoms depends on the extent of the lesion. If general they are universal. If disseminated they are scattered. If transverse they are limited to one level.

Diagnosis of level from situation of symptoms, motor or sensory, see table, page 12.

Symptoms.—(2) Indirect, due to cutting off of impulses to other parts. The cord transmits impulses to and from the brain. In disseminated and transverse myelitis tracts are broken. (1) Motor tracts to parts below, hence secondary lateral sclerosis with its symptoms. (2) Sensory tracts from parts below, hence secondary degenerations upward with their symptoms.

Secondary symptoms are:

1. Rigidity and increased reflexes, contractures, spasms of legs, paralyzed muscles not atrophied, and no R. D.

2. Loss of bladder control without bladder paralysis, priapism.

3. Imperfect sensation without incoördination.

4. Imperfect perception of pain; hence bedsores from dirt, bedsores over sacrum, glutei, heels, and ankles.

Examples: In general myelitis, direct symptoms throughout body.

In disseminated myelitis, direct symptoms in irregular areas, few indirect symptoms.

In transverse myelitis, direct symptoms very limited, indirect symptoms below the lesion.

(*a*) Cervical transverse myelitis, direct symptoms in arms, indirect in legs.

(*b*) Dorsal transverse myelitis, direct symptoms few, in trunk, indirect in legs, arms free.

(*c*) Lumbar transverse myelitis, direct symptoms in legs, arms free, no indirect symptoms.

Course of General Myelitis and Disseminated Myelitis.—Acute or subacute, onset in two weeks after a cold or great strain or after typhus, typhoid, small-pox, confinement, syphilis. Weakness in limbs. Pains in back and limbs. Prostration. Then paralysis. Pain and anæsthesia. Bladder and rectal symptoms. Then patient is confined to bed and bedsores develop. Slow course for a year to three years and occasionally gradual, imperfect recovery. Usually cystitis, bedsores, or pneumonia and death.

Course of Transverse Myelitis.—Acute, onset after injury—blow, fall, wound, dislocation of spine, or strain, causing hemorrhage in cord; or from embolism of spinal arteries.

Chronic, onset after spinal caries with deformity and pressure, or tumor in spinal canal or cord. Course depends on possibility of removing the cause.

Diagnosis.—Myelitis *vs.* multiple neuritis, see page 10.

Myelitis	*vs.*	Meningitis.
1. Very rapid onset.		Slower onset.
2. Fever moderate.		Fever high.
3. Pain in back moderate, in limbs or body dull.		Pain in back and body and limbs very severe, increased by motion.
4. Hyperæsthesia slight in a band at upper level of lesion.		Hyperæsthesia of entire body and limbs very intense.
5. Anæsthesia below lesion.		No anæsthesia.
6. No spasms or rigidity.		Spasms of limbs and back with rigidity of back appear early.
7. Paralysis total in some muscles with atrophy and R. D.		Paralysis only apparent; due to fear of pain on motion; no atrophy.
8. Sphincters paralyzed.		Sphincters not affected.
9. Bedsores and cystitis.		No bedsores or cystitis.

Myelitis	*vs.*	Hysterical Paraplegia.
1. Anæsthesia below lesion includes genitals.		Anæsthesia varies in level and genitals never included.
2. If thighs are paralyzed and atrophied reflexes are lost.		When legs are paralyzed reflexes are exaggerated.
3. Usually some muscles atrophy and show R. D.		Muscles rarely atrophy and R. D. never appears.
4. Sphincters usually involved.		Sphincters rarely involved.

Diagnosis of level of lesion by distribution of anæsthesia, see table, page 12.

Treatment of myelitis.

When onset is acute remove cause, and use counter-irritation, ice-bag, or warm douche. Keep at perfect rest in prone position. Give sedatives and ergot; in early stage give purgatives, calomel, or salts.

When case is chronic in onset or in course:

Employ counter-irritation. Cups, blisters, cautery, ether spray; but not in cases with tendency to bedsores.

Use baths, douches, tepid and cold, to spine.

Keep up strength of patient by good diet, and regulate digestion.

Use massage to the limbs—hot baths for rigidity.

Use interrupted galvanic current to atrophied and paralyzed muscles.

Prevent cystitis by keeping catheters and parts clean and aseptic.

Prevent bedsores by careful padding or water-bed, by sponging with alcohol and alum-water.

Treat cystitis by washing out the bladder, as in surgery.

Treat bedsores by aseptic dressings, as in surgery.

For pain, apply heat or faradic brush to part. Give antipyrin, antifebrin, phenacetine, morphine, salicin, salicylic acid.

For spasms, apply heat to spine, or hot bath to legs, or cups. Give bromides.

For rigidity, when beginning, pads and massage.

For incontinence of urine, pads of absorbent cotton, or wear urinal.

Medicines in chronic myelitis:

Iodide of potash, gr. x. to xx., t.i.d.

— Nitrate of silver in pill with kaolin, gr. ¼ t.i.d. increased.

Ergotin when congestion is suspected.

Strychnine when an increase of reflex activity is not present.

Phosphorus and arsenic as nerve tonics.

Use ung. hydrarg. and KI in large doses in syphilitic cases.

Hemorrhage into the Spinal Cord.

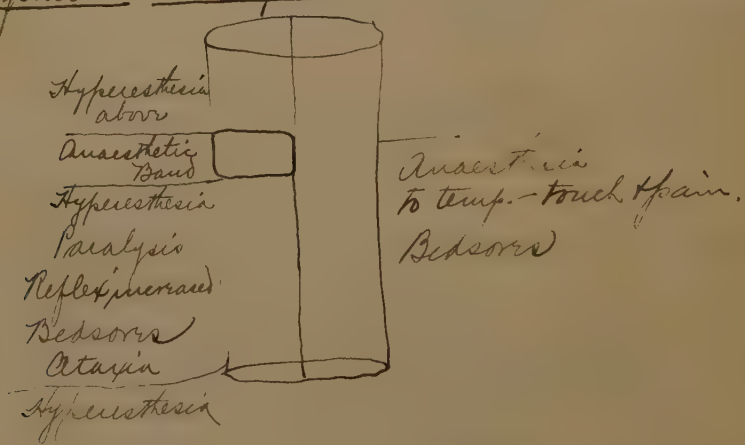
Hemorrhage into the spinal cord or into the membranes is a rare occurrence, and is almost always due to traumatism. The symptoms produced are those of sudden suspension of all spinal functions at

Blisters are dangerous to use.

Avoid morphine as long as possible.
elevate foot of bed.

eflex acts of leg – & control of bladder & rectum
ost up-pain in legs – diff. tumor of
ower cord from sciatica.
f if arm region of cord = spastic gait and
ncrease of leg reflexes and possible sensory
isturbances below.

Brown Sequard's Paralysis: –



and below the level of the hemorrhage. They are therefore identical with those of transverse myelitis, and the sudden onset affords the only means of distinguishing the two diseases. Sometimes spontaneous hemorrhages in the anterior horn cause symptoms of anterior poliomyelitis. Sometimes central hemorrhages cause symptoms of syringomyelitis.

Treat:- Rest Await for absorption or death.

Tumors of the Spinal Cord.

Tumors rarely grow in the spinal cord. They are usually secondary to tumors elsewhere. All varieties may occur. The symptoms produced are those of transverse myelitis of slow onset and characterized by extreme pain at the level of the tumor or in the sensory areas which correspond to the segment in which the tumor grows. The only relief is by operation.

Brown-Séquard's Paralysis.

Hemorrhages in the cord, tumors in or about the cord, and bony growths or callus, the result of injury, sometimes divide or compress one-half only of the cord. The symptoms resulting were first described by Brown-Séquard, and hence the condition has been named after him. This condition is as follows: (1) on the side of the lesion there is paralysis with increased reflexes, and sometimes a loss of muscular sense below the level affected, and a marked hyperæsthesia to touch and pain; (2) on the side opposite to the lesion there is no paralysis, but there is a loss of sensation of touch, temperature, and pain, and sometimes a development of bedsores below the level of the lesion; (3) at the level of the lesion there is a band about the body of anæsthesia, with another band of hyperæsthesia above it; the band on the side of the lesion being a little higher than upon the opposite side.

Concussion or Congestion of Spine:-

After falls & blows - usually no evidence of an ext. injury.

About 1 wk. to 10 ds. after injury:- pains in back - along spine - restlessness - feeling as if desired to stretched or massaged - followed or attended w/ a gradually increasing hyperesthesia to touch of finger - firm pressure w/ hand is not painful - shooting pains in body.

Reflex activity is somewhat heightened.

Sometimes irrit. of bladder.

" flushings of surface - undue sweating - light stroking of the skin is followed by appearance of red streak, wh. widens to about an inch no pain in the streak.

Symps. may gradually extend upward - sometimes also downward. May develop a general neurasthenia. Don't die.

Prognosis:- Recover slowly.

Treat:- Tell him nothing serious is the matter w/ him after impressing him by a thorough examination. Counter-irritation of spine w/ actual cautery - general tonic treatment.

Headaches:-

I. Organic
II. Reflex
III. Circulatory
IV. Toxic V. Neurasthenic. VI. Hysterical

Neuralgia of 5th Nerve & migraine must be separated from it.

Organic occurs in diseases of the brain & meninges. The brain itself is insensitive. Anything producing increased intracranial pressure causes headache.
Meningitis - any form - produces intense headache.
If it is syphilitic - it begins about 5 P.M. increases in intensity & passes off during the night.
Tumors - etc. produce most intense hdache. in the morning after sleep.
Neuritis of the finer nerves produces hdache. all over the head.

Reflex:- Pain referred to ~~irritation~~ head - eye strain above the eyes or back of head or neck. Dull aching hdache. in P.M. - due to use of eyes.
Stomach - indigestion - irrit. of pneumogastric nerve goes to medulla & irritates 5th nerve - usually along line of hair or back of head. Begins in 3 hr. after eating & lasts till digest. is completed.
Irrit. of genitalia - back of head - due to anemia - gonorrhoea - uterine & ovarian dis- chlorosis.

Circulatory:- may be due to too much blood
" " " " " little "
" " " " an alternation of the above

Too much blood - after mental exertion - sides of head
" little " due to anaemia - top " "
Retina shows the condition of the bld. vessels in the brain.

Toxic!- Digestive
Renal
Poisons.

Digestive - improper digestion resulting in auto-intoxication.
Inactive liver - pancreas - intestines.
Treat:- res. digestion - first clean out the patient.
Forehead-bain - give saline if morning
Hair-line " " alone

Renal:- dull - continuous - early if morning -
all over head - esp. forehead.
Treat:- cure Brights!

Poisons!- Infuenza - alcohol - morphine -
chloral - bromide - cannabis indica -
lead - arsenic - mercury.

Neurasthenic:- May be toxic or circulatory -
or reflex or organic.

Hysterical:- imaginary or to a subconscious life X
force themselves on to a conscious life.

Treat:- Reflex - remove cause.
Circulatory improve circulation.
Alternating diet:- rest - exercise -
hot & cold douches - res. digest.
Toxic - treat condition present
Neurasthenic - build up gen. health.

General Remedies for all Kinds of headache:-
~~(Combination of different laxatives - take of 5 or 6 of them)~~
Combination of several drugs.

Antipyrine	gr. XV	Phenacetine	gr. VI.
Sty. Hy. Sulf.	" 1/50	Acetanilid	" III
Caffeine	" I or II	Ignatia	" 1/100

Neurasthenic & Reflex - Ignatia 1/1000 - 4 tablets - every 5 min. until 10 are taken

---

F. E. Guarana
" " Kola } aa q.s. ℥j
" " Celery

Coal-tar-products - better results from one big dose than from repeated sm. doses. Always look for circulatory disturbances & add a drug wh. will compensate for the circulatory failure.

Hysterical headache: - mental in origin - no true pain - may wear out the patient. No remedy for it - only a toxic dose of morphine wh. dulls all senses. The pain is intensified by consciousness & this keeps it up - it is a pain due to forced attention to a part in wh. the pain exists.

Treat: - no medical. By hypnotism.

THE BRAIN AND ITS DISEASES.

I. The Cortex of the Cerebral Hemispheres.

The cortex consists of gray matter spread out in a layer over the cerebral hemispheres, 3 mm. thick and about 200,000 sq. mm. in extent; its amount varying greatly in different persons; its development determining mental capacity. The development of fissures throwing the cortex into folds enables this large extent to be contained in the skull.

The Microscopic Appearance of the Cortex.—Cells arranged in layers.

(1) Superficial or molecular layer ($\frac{1}{1\,0}$) small cells. Much neuroglia.

(2) Layer of small pyramids ($\frac{1}{1\,0}$) with many branches and short process.

(3) Layer of large pyramids ($\frac{4}{1\,0}$) with few branches and a long projection-fibre process.

(4) Layer of polymorphous cells ($\frac{2}{1\,0}$) with many short branches and a short process.

(5) Layer of fusiform cells ($\frac{2}{1\,0}$) with short branches and association-fibre process.

Fibres trasverse these layers, giving off collaterals, and form an intricate network throughout the gray matter. Theré are great variations in the structure of different regions. (See "Quain's Anatomy," 10th Ed., Vol. III. Starr's "Atlas of Nerve Cells.")

The fibres arising from the cortex and constituting the white matter beneath it are (1) association fibres passing to some other part of the cortex; (2) commissural fibres passing to the cortex of the opposite hemisphere; (3) projection fibres passing to the basal ganglia, cerebral axis, or spinal cord.

Divisions of the Cortex into Lobes and Convolutions.—Frontal lobe, 40 per cent.; parietal lobe, 20 per cent.; temporal lobe, 20 per cent.; occipital lobe, 17 per cent.; Island of Reil, 3 per cent. of extent of cortex.

Landmarks on the Cortex.—Fissure of Sylvius, fissure of Rolando, interparietal fissure, parieto-occipital sulcus, calcarine fissure, callosomarginal fissure.

Cranio-cerebral Topography.

To find the fissure of Rolando, lay down a line from the root of the nose to the occipital protuberance over the top of the head and take a point 0.557 of the distance back upon this line. This point will correspond to the upper end of the fissure. The fissure makes an angle of 67° with the median line just measured. Hence if two strips of metal, fixed to one another at this angle, be placed on the head with their junction upon the upper end of the fissure, when one strip is on the median line the other strip, pointing forward and downward, must lie over the fissure of Rolando. In its lower third the fissure becomes a little more vertical than the strip. The fissure is about three and a half inches long.

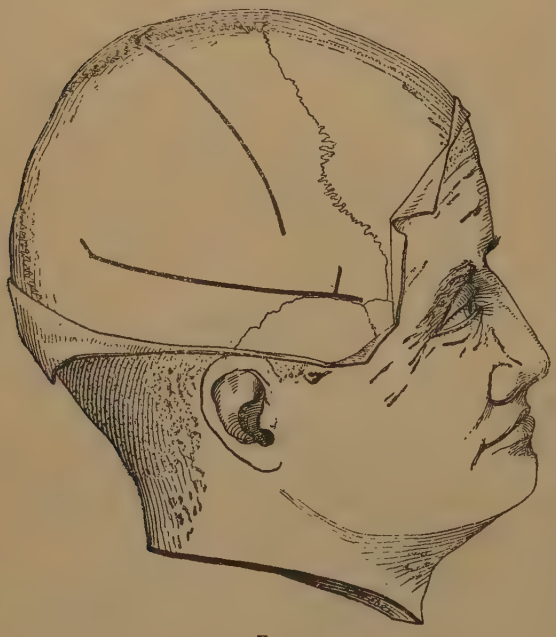


Fig. 4.

To find the fissure of Sylvius, lay down a base line from the lower margin of the orbit to the auditory meatus. Lay down a second line parallel to the base line from the external angular process of the frontal bone backward one inch and a quarter, and then measure upward one-quarter of an inch; this gives point one. Find the most prominent part of the parietal eminence and from it draw a line

downward perpendicular to the base line, and on this take a point three-quarters of an inch below the eminence; this gives point two. Join these two points, and the line will lie over the fissure of Sylvius. The anterior limb of the fissure will be two inches behind the external angular process. The fissure of Sylvius is about four inches long.

To find the parieto-occipital fissure, continue the line of the fissure of Sylvius to the median line. At their junction lies this fissure. Since all areas now open to surgical operation can be located with a definite relation to these three fissures, no further rules are necessary. Since in opening the skull it is customary to make a fenestra of at least an inch in diameter, and it is frequently necessary to enlarge the opening much more, a procedure in no way dangerous under aseptic conditions, there is no difficulty in recognizing the fissures and convolutions exposed if the rules are closely followed. Prior to the large incision of the scalp it is well to mark certain points upon the skull by the sharp point of a chisel, so that when the bone is laid bare surface landmarks may still be kept in view.

Localization of Functions in the Cortex.

Proofs from comparative anatomy; natural and experimental atrophy; process of development; irritation and excision of cortex with various results; pathological lesions of limited extent with secondary degenerations.

The functions of the cortex in general are:

(1) To receive impressions from the various sensory organs as conscious perceptions.

(2) To associate these various simultaneous perceptions into a mental image or concept.

(3) To retain these concepts as memories so that objects can be recognized or recalled.

(4) To give expression to thought in action and in speech.

(5) To restrain the flow of thought and to inhibit or control automatic and reflex acts.

It has been established that various powers of perception, memory, and volition can be definitely assigned to various parts of the brain; that each sense has its corresponding area in the cortex, and each voluntary movement its point of departure from the cortex.

It has been proven that perceptions leave behind them a physical

trace inseparably connected with the area in which they were originally received; that irritation of this area produces a renewal in consciousness of the original perception as a memory, which may be so vivid as to appear real, being then a hallucination; that destruction of this area causes a loss of the power of recalling the previous perception or of recognizing it when repeated.

It has been proven that movements which are acquired by practice, and are therefore the result of conscious effort, are always initiated from a definite area; that this area is inseparably related to movements; since its irritation, by any cause, results in the production of forcible movements of the nature of spasms, and its destruction produces a loss of the power of movement or paralysis of voluntary action.

It has been proven that the use of language involves both the recollection of auditory and visual symbols and the initiation of impulses of speech and writing, thus calling into play not only the motor portion of the brain, destruction of which therefore causes a loss of speech, but also the sensory portion of the brain, a lesion of which will also cause aphasia.

There are certain areas upon the cortex of the brain, not necessarily coextensive with either lobes or convolutions, whose functions are accurately known.

These areas are: (1) The sensori-motor area. (2) The visual area. (3) The auditory area. (4) The area of sensations of smell and of taste. (5) The speech areas.

The Sensori-Motor Area.

The sensori-motor area includes the cortex of the anterior and posterior central convolutions which border the fissure of Rolando and the adjacent cortex in front and behind these convolutions. Each hemisphere controls movement on the opposite side of the body, but as the right hand is more generally used and is better trained than the left, this area is larger on the left hemisphere than on the right.

The cortex of the posterior part of the second frontal convolution controls the movements of the eyes and head. Impulses starting from this area produce conjugate movement of these parts toward the opposite side. The eye district is below, the head district above.

The lower third of the anterior and posterior central convolutions governs the movements of the face, tongue, larynx, and pharynx.

The eyebrows and cheeks are controlled by the upper and forward part of this area; the tongue and larynx by the lower and forward part; the mouth, pharynx, and platysma by the hinder part.

The middle third of the anterior and posterior central convolutions governs the movements of the upper extremity; the shoulder motions being controlled in the anterior and upper part of this area, the elbow motions in its middle part, and the hand and finger motions in its posterior and lower part.

The upper third of the anterior and posterior central convolutions, including their junction in the paracentral lobule, controls the motions of the lower extremity, the thigh, knee, foot, and toes being governed by various parts of this area from before backward in the order named.

The parts susceptible of the finest and most delicate movements, those directed by the most acute sensations, the lips, the fingers, and the toes, lie furthest back in the motor area, chiefly in the posterior central convolution. Lesions in this convolution almost always cause some loss of tactile sensation as well as paralysis, and hence this area is thought to be the seat of tactile sensations as well as of movements, while some cases point to the localization of muscular sensations in the area just behind that of motion.

The median surface of the hemisphere in front of the paracentral lobule is known to be related to movements of the trunk in monkeys, but the cortex in front of the leg area on the convexity is the probable location of the trunk area in man.

There are no sharply defined sections of the motor area to be assigned to special motions. Each motion, each part of a limb, has a wide general representation over the cortex and a special representation at a limited area. The areas of representation of different limbs merge into one another; thus, in the representation of the thumb we find that there is a focus, but that the thumb is represented over a great deal of the upper limb region, and that this representation diminishes in intensity gradually as we pass from the focus upward. This explains the fact that the excision of a small area does not totally paralyze the portion of the limb represented chiefly on that area. The adjacent areas represent to some extent that limb, and hence can govern it if need be.

The motor centres govern motor acts rather than special muscles, each act involving a succession of movements of different joints by various groups of muscles.

Cortical Spasm and Paralysis. — Irritation in the motor area produced by any kind of disease gives rise to localized spasms or convulsions, called Jacksonian epilepsy. Destruction of the motor area gives rise to paralysis. The point of beginning of the spasm or the extent of paralysis depends on the part of the motor area diseased. Hence, from the character of either of these symptoms a conclusion can be reached as to the location of the disease. Cortical spasm or paralysis usually begins in the face or in one limb of one side and extends. The order of the progress also indicates the location of the disease, *e.g.*, from face to arm, then to leg; or *vice versa*, *e.g.*, spasm of shoulder, arm, and hand, *vs.* spasm of fingers, arm, and shoulder: the disease is most intense in the part first affected. Sensations of numbness precede the spasm. A cortical spasm begins locally, and usually extends until one side of the body is affected. It is usually followed by temporary weakness. Consciousness is preserved.

Cortical paralysis is monoplegic in type *vs.* subcortical paralysis, which is hemiplegic in type. A slowly advancing paralysis indicates a slowly extending destruction of tissue. Cortical paralysis is associated with increased reflexes, but there is no atrophy and no change in electric reactions. Contractures develop late.

The Visual Area.

The area of sensations of sight is located in the occipital lobe of the brain, including the cuneus on the median surface and the occipital convolutions on the convexity. The cortex lying in the calcarine fissure is the part primarily reached by the visual impulses, but the parts named are also concerned in vision. Each occipital lobe receives impressions from one-half of both eyes; hence a lesion in one lobe produces hemianopsia, a half-blindness in both eyes, the blind field of vision being on the opposite side to the lesion. This is termed homonymous hemianopsia; that is, a blindness in the like-named halves of both visual fields. When the right occipital lobe is affected, the patient cannot see anything on the left side of the middle line as he looks forward.

Irritation of the occipital lobe may cause hallucinations of light or even of objects which are referred to the opposite visual field. Destruction of the occipital lobe on the left side destroys sight-memory pictures; hence recognition of things seen may be impossible, and reading power may be lost.

The Auditory Area.

The area of sensations of sound is located in the first and second temporal convolutions of the brain. Each ear is connected with both hemispheres; hence deafness from a unilateral lesion is only partial and is not generally noticed. But if both temporal lobes are destroyed the patient becomes totally deaf. Irritation of the temporal cortex may cause hallucinations of hearing. This may be the first symptom of a convulsion whose origin is an irritation in this part of the cortex. Destruction of the temporal cortex on the left side causes word-deafness. (See speech areas.)

The Smell and Taste Area.

The area of sensations of smell and taste is located at the tip of the temporal lobe on its under and inner surface, which rests on the sphenoid bone. Each lobe is related to sensory organs on both sides, and unilateral lesion does not often produce noticeable symptoms.

The Speech Areas and Aphasia.

The speech areas are of four kinds and in four locations. They are limited to the left hemisphere in right-handed persons and to the right hemisphere in left-handed persons. There is *the motor-speech area* in the posterior part of the third frontal convolution (Broca's convolution), in which the movements concerned in the act of speaking are controlled. The use of language and the power of talking are affected when this region is destroyed. There is the *auditory-speech area* in the first and second temporal convolutions, in which the memories of word-sounds are stored up. The understanding of language and the power of recollecting the names of objects are lost when this region is destroyed. There is *the visual-speech area* in the occipital and lower parietal region, in which the memories of printed words are stored up. The understanding of written language and the power to read are lost when this region is destroyed. The *power of writing* is a part of speech and is usually lost when the motor-speech area is destroyed, but its exact location is not fully determined; some cases pointing to the second frontal convolution, others to the lower parietal convolution near the hand centre as its probable cortical position. (See Fig. 5.)

It is accepted that every word in ordinary use has a complex mental substratum, which may be termed the word-image, made up of a number of memory-pictures. The memory of the sound of a word as spoken, the memory of the appearance of the word as printed, the memory of the muscular movements needed to write the word or to pronounce it, are known to be distinct from one another and yet to be associated together. Loss of one of these memory-pictures, or

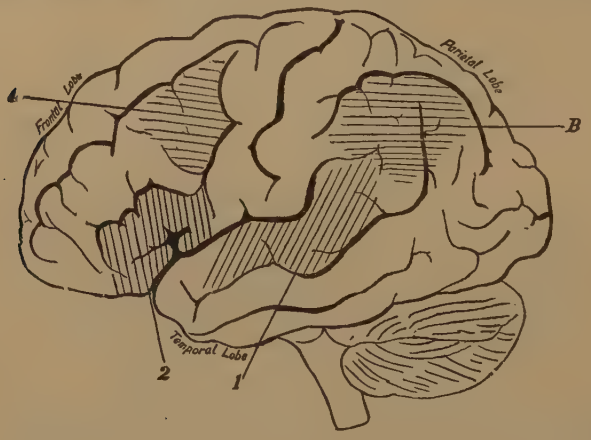


FIG. 5.—The Speech Areas: 1, auditory area; 2, motor area; B, visual area; 4, writing area.

disturbance in their asssociation, impairs the integrity of the word-image, and produces such defects in its use as are indicated by the names given to the respective varieties of aphasia. A division of disturbances of speech has been made into two great classes of sensory and motor aphasia—the first due to defect in the receptive, and the second due to impairment of the emissive functions of the brain. A further subdivision into several varieties has been made. These are word-deafness, word-blindness, agraphia, motor aphasia, and paraphasia. But, before considering these defects, let us look at the physical basis of the thought which lies back of the word. Take the idea "bell" as an example.

The idea of a bell is made up of separate memory-pictures of its shape, size, color (visual-picture); weight (muscular-sense picture); cold smooth surface (touch-picture); and sound (auditory-picture). These various memories, received by different senses and stored up in different parts of the cortex, are associated together in a mental image of the object bell.

To this mental image the word-image is added of the word bell as heard, as printed, as spoken, as written, making up a congeries of memory-pictures forming a "concept." Any one of these memory-pictures may be lost by disease. If the memories of the object are lost the symptom is called *apraxia*. If the memories of the word are lost the symptom is called *aphasia*.

To examine an aphasic thoroughly it is necessary to test—

1. The power to recognize objects seen, heard, felt, tasted, or smelt, and their use.

This will determine whether the condition of apraxia is present.

2. The power to recall the spoken name of objects seen, heard, handled, tasted, or smelt.

3. The power to understand speech and musical tunes.

4. The power to call to mind objects named.

This will test the integrity of the auditory-speech area and of the association-tracts between other sensory areas and the temporal convolutions.

5. The power to understand printed or written words.

6. The power to read aloud and to understand what is read.

7. The power to recall objects whose names are seen.

8. The power to write spontaneously, and to write the names of objects seen, heard, etc.

9. The power to copy and to write at dictation.

10. The power to read understandingly what has been written.

These tests will determine the condition of the visual word-memories in the angular gyrus, and of the connections between this area and surrounding sensory and motor areas.

11. The power to speak voluntarily, and if it is lost, the character of its defects.

12. The power of repeating words after another.

This will test the integrity of Broca's centre and its association-tracts.

1. *Word-Deafness.*—If the memory of the sound of the word is lost, the word cannot be called to mind and cannot be recognized when heard. Show the patient a watch, and he is unable to name it; tell him it is a stone, a match, a watch, and notice whether he dissents from the former and gives signs of satisfaction at the last. If he does, he has only auditory amnesia, but not word-deafness. If not, he is word-deaf, and is unable to understand what is said to him. The lesion lies in the posterior half of the first and second

temporal convolution in the left hemisphere in right-handed, in the right, in left-handed persons. This is one variety of aphasia whose lesion is well known, though it rarely occurs alone.

2. *Word-Blindness.*—If the memory of the appearance of the word is lost, the visual image of it cannot be called to mind or recognized, and then the patient will be unable to write spontaneously, for he cannot remember how the letter looks which he wishes to write; and he will be unable to read, because the shapes of the letters and words seen arouse no recollection. As a matter of fact, words are forgotten more easily than letters, and if a patient is to relearn to read he must begin with letters and go on to words. Figures are sometimes recalled when words are forgotten, and many a patient can do mathematical calculations on paper who cannot read or write ordinary words. The reverse may also be true, the patient being able to read and write, but being unable to understand figures or calculate. Such patients may also play cards or other games, if they are not psychically blind. It is not infrequently the case that persons who are thus word-blind can write at dictation, or copy, and yet show no evidence of understanding what has just been read or written. A distinction must be made between those who can and cannot do these things, though its pathological basis is still obscure. The condition of visual amnesia with word-blindness is due to a lesion involving the inferior parietal convolutions and angular gyrus, and is often associated with psychical blindness, but may occur independently of it. Word-deafness and word-blindness frequently occur together, and then the lesion is found involving both the temporal and the angular convolutions. Some cases of this condition have depended on a deep lesion in the temporo-occipital region, breaking the association-tracts.

3. *Motor Aphasia.*—If the memory of the effort needed to pronounce a word is lost, a true paralysis of active speech occurs, though the muscles may not be weakened. This is the ordinary form of motor aphasia, due to a lesion of Broca's centre. It is to be noted that such a loss of speech involves a loss of the power of repeating words after another, as well as of voluntary speech, and is not accompanied by any inability to understand spoken or written language. In the uneducated, as in children, the acts of talking and writing are closely joined, as may be seen by watching the lips, which move in the act of writing. But among those accustomed to write much these acts are independent, and it is probable that many

educated aphasics may be able to answer questions in writing when their efforts at speech fail. Reading aloud will also be lost in motor aphasia, for here, too, the inability to articulate hampers the patient.

4. *Agraphia.*—The independence of the effort-memory necessary for writing, and connected with movements of the hand from the effort-memories of speech, has been alluded to. When these are lost alone the condition is known as agraphia. In such a state the pen cannot be used. Copying, writing at dictation, and voluntary writing are all lost. It has been noted already that when a word cannot be called to mind, it of course cannot be written. But words can often then be written at dictation, if the person is one who writes much. Hence sensory agraphia and motor agraphia must be distinguished; the former being a part of word-blindness, the latter not at all associated with inability to read. The lesion of motor agraphia is unsettled, though a few facts point to the posterior part of the second frontal convolution as the probable seat of this function. It is not unlikely, however, that the more exact localization of fine movements of the thumb and fingers in the posterior central convolution by Horsley may be followed by the discovery of the writing centre in this vicinity.

5. *Paraphasia.*—These forms of aphasia are due to a loss of distinct memory-pictures. The several memory-pictures which are united in the word-image may thus be reasonably regarded as separate from one another in their location in the brain. But since they *are* joined together to form the word-image, it follows that the association-fibres joining the various areas are as necessary to the use of even a single word as the various areas with their memories. It is really by association only that an object or a word becomes a subject of thought or use. If these associations are broken, the result is a defect of language characterized by the misplacement of words, and the patient talks jargon.

There are many forms of paraphasia as there are many association-tracts, and it is not yet possible to assign different cases to their lesions.

It is evident that many differently located lesions will produce disturbances of speech. When the entire cortex is diseased—as in paresis—aphasia also appears.

Defects in the appreciation and use of music are called amusia, and present the same varieties as aphasia.

The Frontal Region.

There appears to be a certain relation between the frontal lobes of the brain and the higher forms of intellectual activity, the powers of fixing the attention and of reasoning and of self-control. But disease here does not cause a loss of any one mental faculty, and for the higher powers of the mind a general integrity of the entire brain, not of any one part, is necessary. When it is considered that every concept is made up of numerous memory-pictures joined together, each of which has a separate location in the brain cortex, it becomes evident that to the process of thought a healthy state of the entire cortex is necessary, and also of the white matter beneath it, through which the associating fibres pass. And it is therefore impossible for a single lesion anywhere to cause a loss of memory or of imagination or of judgment. Yet for the co-ordination of facts into orderly series, for comparison, and for analysis of knowledge gained through the senses, the healthy state of the frontal lobes appears to be necessary. And lesions in the frontal region, especially upon the left side, are quite uniformly attended by mental dulness, apathy, lack of power of concentration, and imperfect self-control.

There are large areas of the cortex of the brain whose function is undetermined. These are much more extensive on the right hemisphere than on the left. There are no definite symptoms produced, so far as we now know, by lesions in these areas; but the negative fact is certain, that lesions in them do not cause disturbances of motion, of sensation, or of speech.

The cortex of the hemispheres upon the base of the brain lying on the orbital plate, on the sphenoid and temporal bones, and on the tentorium cerebelli, has as yet no assignable functions, and lesions in these regions do not produce recognizable symptoms.

Symptoms of Cortical Disease.

In the study of cases of cortical disease, it is necessary at the outset to distinguish general from local symptoms, for it is the latter only which enable one to locate the lesion. *General Symptoms* are those which are common to various kinds of brain disease in whatever region they are located. They are headache, vertigo, digestive disturbances, general convulsions, optic neuritis, with or

without blindness, delirium, and coma. These are due either to an increase of the intracranial contents (as by the growth of a tumor) and consequent pressure, or to some other interference with the normal condition in the brain. They give no evidence as to the exact region of the brain which is affected. They merely indicate that the brain is diseased.

Local Symptoms, on the other hand, depend entirely for their production upon the region of the cortex which is invaded. They are disturbances of motion and of sensation of various kinds, and of sensory perception; disturbances of memory; and loss of speech. They may be divided into symptoms of irritation and symptoms of destruction; and it is not infrequently the case that the former precede the latter in the course of a disease. An irritative lesion produces symptoms due to an increased activity of the area affected; *e.g.*, spasms, pain, tingling and numbnesss, flashes of light, sounds, or hallucinations of smell and taste. A destructive lesion produces symptoms due to a loss of function in the area involved; *e.g.*, paralysis, anæsthesia, blindness, deafness, loss of smell and taste, and of the use of language. Each of these points to a different location of the disease, and is essential to its localization.

II. Tracts within the Brain.

The Association-Tracts.

The first system of fibres in the centrum ovale is the *association* system.

It can be shown by careful dissection that each convolution is joined to the two adjacent convolutions by fibres which pass around the separating fissures. Also, that bundles of fibres exist which pass from each convolution to the convolution next but one, and so on. Hence, it may be stated that each convolution has a possible connection with every other. Besides this association of convolutions by small bundles of fibres, it is possible to find a distinct set of association-tracts which pass between more or less distant regions. One such tract passes from the frontal lobe, collecting its bundles from all three convolutions, backward to the occipital lobe. Another tract joins the occipital with the anterior part of the temporal lobe. Another passes from the upper two temporal convolutions forward to the third frontal convolution, passing beneath the island of Reil.

And a tract from the frontal to the posterior temporal area may also be found.

The function of these association-fibres is to form the physical basis for the association of concrete memories and of psychical acts, and their integrity is necessary to thought. By studying subjectively the association of ideas in the mind the importance of their function is evident. When they are destroyed by disease various forms of paraphasia appear.

The Commissural Tracts.

The second system of fibres in the centrum ovale is the *commissural* system. This joins corresponding areas of the two hemispheres with one another. The commissural fibres between the frontal, parietal, and occipital lobes of the two sides pass in the corpus callosum. Those from the temporal lobes pass in the anterior commissure. The function of these fibres is to harmonize the action of the two hemispheres. Simultaneous movements of like nature can be made with great facility with both upper extremities. Movements which are difficult when attempted with the left hand alone become easy when associated with corresponding movements of the right hand—as, for example, drawing a circle, writing one's name. It is the commissural fibres joining the motor convolutions only which can be thus tested. Failure to perform easily corresponding bilateral motions in face, hands, or feet would indicate some obstruction to conduction in them.

The Projection-Fibres.

The *projection* system includes those fibres which join a definite area of the cortex with parts of the nervous system lying below it, viz., some nervous mechanism in the basal ganglia, brain axis, or spinal cord. Indirectly, through the medium of such mechanisms, the external world is projected upon the brain and reaches consciousness, and voluntary impulses originating in the brain are sent to the muscles. These fibres gather together within the hemisphere at the upper level of the basal ganglia, and either end in the optic thalamus or go on between these ganglia through the internal capsule to the brain axis and spinal cord.

The majority of these fibres end in the optic thalamus, which is thus connected with all parts of the cortex of the brain. Of the

function of these we know very little. Two large bundles, however, are separable from the mass.

The Visual Tract.—One of these passes inward and forward from the occipital lobe, and joins the pulvinar of the thalamus and the external geniculate body. This is *the visual tract.* (Fig. 6, 4.)

The optic nerves decussate partially at the chiasm, so that each optic tract contains fibres from both retinæ. Each tract passes to the optic thalamus, external geniculate body, and anterior corpus quadrigeminum. Thence the fibres of the visual tract pass out, to turn backward through the internal capsule and through the centrum ovale to the cortex of the occipital lobe. A lesion anywhere in this course causes homonymous hemianopsia.

Fig. 6.

The Auditory Tract.—A second bundle passes from the temporal lobe upward to the thalamus, internal geniculate body, and posterior corpus quadrigeminum, in which the auditory tract from the acoustic nucleus ends. This conveys impulses of sound from both ears to each temporal lobe, and is the *auditory tract.* These are the only bundles of the thalamic radiations whose function is determined. (Fig. 6, 9.)

Some of the projection-fibres pass on through the internal capsule without communicating with the basal ganglia, and of these we know three distinct bundles. (Fig. 6, 1, 2, 3.)

The first is collected from the three convolutions of the frontal lobe, and passing between the caudate and lenticular nuclei in the anterior division of the internal capsule, descends in it to the base of the brain, and issuing in the inner third of the foot of the crus

cerebri, passes down to the pons, where it terminates in nuclei lying in the ventral half. The nuclei thus reached by these fibres are also joined by other fibres from both hemispheres of the cerebellum, which enter the pons at its lateral surfaces in the middle peduncles. Thus it is evident that a connection exists between each frontal lobe and both cerebellar hemispheres, the crossed connection being greater than the direct one. Of the function of this tract we know nothing.

The Motor Tract.—The second bundle of the projection system is *the motor tract.* It comes from the posterior part of the third frontal convolution, the two central convolutions, and from the paracentral lobule, and passes out of the base through the middle third of the crus cerebri. Its fibres collect at the middle portion of the upper surface of the internal capsule, those from the lower parts of the cortex passing straight inward, those from the upper parts curving outward and downward to pass around the side of the lateral ventricle. Thus within the centrum ovale, these fibres, if looked at from in front, appear like the sticks of a fan, and like those sticks their relative position is altered in the point of junction, where those passing inward from the lowest part of the cortex lie in front of those which pass downward from its upper part. Thus in the capsule the order from before backward is, *first*, the fibres conveying speech impulses to the pons and medulla; *second*, the fibres conveying facial-motor impulses to the pons; *third*, the fibres destined to the arm centres of the cord; *fourth*, the fibres transmitting impulses to the leg centres in the cord. The fibres conveying impulses to the muscles of the trunk probably lie behind those to the leg.

From the anterior half of the posterior division of the capsule this tract passes through the middle third of each crus, through the pons (where the division to the facial nucleus crosses to the opposite side and ends), and thence by way of the pyramids of the medulla to the crossed pyramidal and direct anterior median columns of the spinal cord. It is evident, however, that the concentration of this tract is much greater in the capsule than in the centrum ovale, where the individual fibres are scattered among the other systems and occupy but a small area from before backward.

Lesions beneath the third frontal convolution of the left side produce motor aphasia. Lesions beneath the central convolutions in the centrum ovale produce paralysis, which will vary according to the position of the lesion. The nearer the lesion lies to the cortex the more will the symptoms resemble those of cortical disease,

monoplegia being the rule. The nearer the lesion to the point of junction at the capsule the more will the symptoms resemble those of capsule lesion, hemiplegia being the rule. When the motor tract is injured by a lesion in the lower half of the pons, the result is paralysis of the face on the side of the lesion and of the arm and leg on the opposite side (alternating paralysis).

The Sensory Tracts.—The third set of fibres of the projection system includes those which lie just posterior to the motor tract, and which pass inward from the parietal convolutions. These take a similar course to those of the motor tract, and fill up to a considerable extent the space between it and the radiation of the visual tract, toward the occipital lobe. They are mingled with fibres which pass to the optic thalamus, but are separable from them in fœtal brains, and may be traced down through the capsule to the tegmentum of the crus, where they divide into a portion going to the lemniscus, and a portion going to the formatio reticularis. The fibres can be traced through these tracts downward to the medulla and through the sensory decussation to the posterior pyramids of the medulla in which the posterior columns of the spinal cord begin. This set of fibres conveys the sensations of touch, pain, temperature, and muscular sense, and lesions in its course will cause disturbance of these sensations. Like lesions in the motor tract, the rule obtains that the nearer the cortex the more likely is the lesion to cause an affection of a single limb, while the nearer the capsule the more likely is the symptom produced to be hemianæsthesia. A lesion in the lower half of the pons may produce alternating hemianæsthesia, the face being anæsthetic on the side of the lesion, and the limbs on the opposite side.

From these facts it is evident that a lesion which lies in the centrum ovale, at any point posterior to the præcentral fissure of the frontal lobe, may produce recognizable symptoms, for it must affect either the motor, or the sensory, or the visual, or the auditory tracts, or individual fibres of those tracts.

III. The Basal Ganglia.

The symptoms produced by lesions in the basal ganglia—corpora striata and optic thalamus—are not to be distinguished from those caused by lesions of the projection-tracts which lie near them.

Lesions of the corpora quadrigemina cause nystagmus, blindness, and ophthalmoplegia (see page 17).

IV. The Cerebellum.

The cerebellum is the central organ of equilibrium. It is joined to the spinal cord by the direct cerebellar column, which carries sensory impulses up to it through the inferior peduncle. It is joined to the motor tract by the middle peduncle, which conveys impulses to the pons and thence to the spinal cord, directing balancing motions. It is joined to the cerebral hemispheres both by the middle and superior peduncles, whose exact functions are unknown.

Lesions of the cerebellum, or of its peduncles, cause staggering gait. This differs from that in locomotor ataxia, for there is no ataxia of the limbs when the patient lies down, giving support to the body. When the middle peduncle is irritated the patient staggers toward the lesion; when it is destroyed he staggers away from the lesion. Lesions of the cerebellum also produce vertigo.

The Cerebral Diseases of Vascular Origin.

The most common cerebral diseases are those due to a rupture of a vessel in the brain, *cerebral hemorrhage*, or to a stoppage in a cerebral vessel, *cerebral embolism* and *cerebral thrombosis.*

The cerebral circulation. See "Quain's Anatomy."

Pathology. — The condition which leads to such diseases is chronic endarteritis; a chronic inflammatory process in the intima and muscular coats of the vessels with a production of new cellular tissue which may (1) go on until it obliterates the lumen of the vessel, producing thrombosis; or (2) undergo calcareous degeneration, roughening the wall and inducing fibrin deposits, which may occlude the vessel, producing thrombosis, or be washed onward, producing embolism; or (3) undergo fatty degeneration, thus eroding the intima and weakening the muscular coat, causing aneurisms (miliary or larger) which rupture, producing cerebral hemorrhage. See Delafield and Prudden's "Pathology."

Pathology of Cerebral Hemorrhage.—The vessel ruptures. The blood spreads out in the membranes, filling the fissures and depressing the cortex, or if the hemorrhage is subcortical, tears the surrounding brain. A clot forms, thin and flat on the cortex, globular in the brain, which compresses the brain-tissue around it. This clot gradually shrinks and undergoes absorption, leaving a hæmatin stain; the disintegrated brain-tissue undergoes fatty degeneration and absorption. There remains a mass of softened tissue without or with

a connective-tissue wall, or finally a cyst, or a mass of sclerotic tissue or merely a scar. The torn brain never reunites. From the point of laceration degeneration begins, which extends along the various tracts, *e.g.*, descending degeneration in motor tract through the cerebral axis and spinal cord, or ascending degeneration in sensory tracts to the cortex. When the pressure is removed the cortex may resume its function or the uninjured tracts may resume their function. Hence partial recovery is the rule after cerebral hemorrhage.

Pathology of Cerebral Embolism or Thrombosis.—The vessel being occluded, the brain-tissue which is supplied with blood through it is cut off from its nutrition. The first arrest of blood supply is extensive, but when collateral circulation is established the final area or region which softens may be small. In the cortex the collateral supply by anastomosing vessels is extensive. In the brain ganglia and capsule it is very imperfect. Hence the permanent effect of occlusion is more serious in the arteries entering the base than in small branches in the cortex. If large vessels in the cortex, *e.g.*, a main branch of a Sylvian artery, is plugged, the area of softening may be extensive. A fatty degeneration and a necrosis of brain-tissue follow the occlusion of the vessel. In the softened tissue there are at first extravasations of venous blood (red softening); later these are absorbed and the tissue in a stage of fatty degeneration is yellow (yellow softening). If there is simple necrosis without extravasation or fatty degeneration, the brain has its natural color but is soft (white softening). The softened tissue contracts, leaving a depression, or a cavity, or a scar; in some cases a cyst with smooth walls and serous contents.

The Situation of the Lesion.—Cerebral hemorrhage: in basal ganglia involving the internal capsule in fifty per cent., next in frequency in centrum ovale, cortex, pons, and ventricles; very rarely in cerebellum.

Cerebral embolism, forty-nine per cent. left; forty-one per cent. right; ten per cent. both hemispheres. Central ganglia and capsule, sixty per cent.; next in frequency cortex supplied by middle cerebral artery, (1) motor area, (2) aphasic area, (3) visual area. Least frequent pons and anterior or posterior cerebral arteries.

Cerebral thrombosis in middle cerebral, basilar, vertebral, and anterior and posterior cerebral vessels in order of frequency named.

Etiology.—Thrombosis is common below age of forty-five; hem-

orrhage after forty-five; age of maximum liability to hemorrhage, sixty (~~to seventy~~). Males more liable than females to hemorrhage. Predisposing causes of endarteritis are gout, lithæmia, alcoholism, and syphilis; associated diseases are nephritis, endocarditis, and emphysema. Anything which increases the heart action may cause cerebral hemorrhage, *e.g.*, fright, anger, exertion, cold bath. Anything which weakens its action may cause thrombosis.

Symptoms.—Preliminary: For months before the onset, vertigo, insomnia, headache, inattention, and imperfect memory. Temporary attacks of numbness in one-half of the body.

Sudden onset, "apoplexy," loss of consciousness. The result depends on the size and rapidity of hemorrhage, or on the size of the vessel occluded.

Paralysis of one-half of the body (hemiplegia), with coma and convulsions, is the ordinary form. Face, red; pulse, slow, high tension, full; respiration, deep, stertorous. Temperature normal, but after some hours high, 102° to 104° F. Deviation of eyes and head may occur in large lesions; the patient looks toward the side of lesion. Pupils vary. Inequality is most important symptom, usually dilated on side of lesion. Hemiplegia, hemianæsthesia, and hemianopsia, at first. Reflexes suspended; inhibited by irritation for a short time, then exaggerated permanently. Rigidity of limbs when hemorrhage is large or into the ventricle; convulsion when hemorrhage is on cortex or in ventricle. Urine retained; contains albumin and sugar, or may be passed involuntarily.

Death may occur in one or two days, in coma, with temperature of 108° F.

Usually there is a slow recovery of consciousness and permanent paralysis, which gradually decreases during a year, and then remains stationary.

In lighter cases there may be no loss of consciousness, but partial hemiplegia or arrest of any cortical function.

The permanent symptoms a week after the acute onset indicate the situation of the lesion. The symptoms which remain at the end of a month indicate its extent; *e.g.*, hemiplegia with hemianæsthesia and hemianopsia at first; and permanent paralysis, not total, in arm and leg only, without sensory affection finally. In some cases there is a development after two months of contractures in the paralyzed limbs. This indicates the existence of descending degeneration in the motor tract.

Pressure across front of head or pain in back of head.

Dilatation of pupil on side of lesion occurs only if hemorrhage, due to increased intra-cranial pressure.

Diff. Diagnosis of Coma.

Epilepsy:- History of convulsions
may be blood & froth at mouth
Can get response from limbs.

Uraemia:- Contraction of pupils
Foul breath
Odor of urine
oedema of extremities

Diabetes:- History of case
Sugar in urine

Alcohol:- Dilated pupils
Smell of breath

Opium:- Simulates hem. & tox coma
Respir. slow
Pupils contracted

Sun Stroke:- Diffusion of face
Exposure to heat
Reflex not changed of either side

Cerebral Injury:-
Same as apoplexy.

Course.—After the acute onset, in which the patient is confined to bed for two weeks to two months, depending on the severity, the improvement begins; the chief symptom being hemiplegia. The face is flat on the paralyzed side, and is slightly drawn by the healthy muscles away from the paralyzed side so that the mouth is crooked. The eye can be closed (*vs.* facial palsy), and can be moved (*vs.* ophthalmoplegia). The tongue deviates toward the paralyzed side. The finer motions of the hand and arm are more permanently affected than elbow or shoulder motions; and the upper extremity is carried in a flexed position. The leg is stiff and extended, does not bend easily at the knee, and the foot is dragged on its inner edge and swung around in walking. The muscles of the trunk and respiration are very rarely affected. The bladder and rectum may not be under full control. There may be a diminution of sensation in the paralyzed side, and possibly hemianopsia. Hemianæsthesia or hemianopsia may remain without hemiplegia. A total loss of all senses, smell, taste, sight, hearing, and touch on one side only, occurs in hysteria only. The tendon reflexes are usually much increased on the paralyzed side, and are somewhat increased on the other side as well. The contractures which develop may cause pain, because the limbs are rigid. Otherwise there is no pain.

Aphasia of some kind often accompanies right hemiplegia, or may occur alone. (See page 49.)

Some mental symptoms are usually observed after an apoplexy, and may be the only permanent symptoms. There may be a lack of control of the emotions, so that the patient laughs or cries with little cause; an irritability of temper; a lack of judgment; imperfect memory of recent events; imperfect power of concentrated attention; general bewilderment, so that the patient does not recognize his surroundings or friends; mild delirium or delusions; or even dementia. These usually pass away gradually, but sometimes remain.

The various symptoms, motor, sensory, aphasic, or mental, depend on the situation of the lesion, and enable a conclusion to be arrived at regarding it.

In any case epileptiform attacks may develop, and continue at long intervals after an apoplexy.

Diagnosis. Cerebral Hemorrhage.—In old persons with atheromatous arteries and hypertrophied heart. Sudden onset of coma during exertion or in excitement. Coma deepens; temperature falls

to 97° to 95° F. in an hour; after three hours rises to 101° F. Gradual recovery of consciousness in three to five days, with permanent symptoms of hemiplegia.

Cerebral Embolism.—At any age with heart disease, or after childbirth. Sudden onset with no loss of consciousness or slight condition of mental confusion, or rapid return of consciousness. Temperature does not fall, but may rise to 102° F. Improvement occurs within twenty-four hours to a marked degree, but symptoms return in three or four days and are permanent, either monoplegia, or hemiplegia, or aphasia. Jacksonian epilepsy may develop after a few months.

Cerebral Thrombosis.—At any age, but chiefly in syphilitic persons and in middle-aged men. Premonitions usually occur. Slower onset without coma, but dulness of mind. Consciousness returns soon, if lost. Temperature does not fall; may rise to 100° F. The paralysis resembles that in embolism.

The diagnosis between these three conditions is never positive.

Causes of coma other than apoplexy: Syncope, epilepsy, uræmia, diabetes, alcoholism, opium poisoning, sunstroke, cerebral injury. Apoplectic coma is deep and patient cannot be aroused. Face is congested, eyes turned to one side, pupils unequal, paralysis is unilateral, limbs may be rigid; appearance indicates previous convulsions. Physical examination reveals absence of injury and presence of predisposing disease, *e.g.*, atheroma, endarteritis, cardiac disease, albuminuria, syphilitic scars. The absence of diagnostic signs of other causes of coma confirms the diagnosis of apoplexy. Reflex movements can be caused on non-paralyzed side by producing pain. The tendon reflexes are often lost at the onset. The cremasteric reflex is always lost on the paralyzed side.

Prognosis depends on the depth of coma, on the condition of pulse and respiration, on the constitution and age of the patient, and on the number of previous attacks.

Treatment in stage of apoplexy: Rest; quiet; ice-bag to head; venesection in plethoric cases; purgatives of a drastic nature; stimulants if heart failure is present.

For hemiplegia: Exercise; massage; faradism to the muscles for purpose of exercise.

Medicines are of no use in either stage.

F. Nitroglycerin if embolus is suspected, to dilate vessels & carry it further.

Cremasteric reflex is absent on the paralysed side. This is a valuable diff. point in hysterical paraple

Treat. Put ice-bag to head.

In plethoric pts. w/ vigorous heart action bleeding often does good.

Purge by croton oil - putting it on butter in side of mouth.

Turn patient on side, esp. when there a bounding pulse & stertorous breathi in coma, this throws the tongue forward & keeps the larynx open & prevents CO2 poisoning. Always place paralysed sid up, so that lesion is down then orhage if it burrows will go outw. and not

Cerebral Atrophy.

Cases of cerebral disease in children, either unilateral or bilateral, present some peculiar features of three different types.

First, cases of hemiplegia with or without athetosis.

Secondly, cases of mental defects of various grades.

Thirdly, cases of sensory defect of different types. Epileptiform seizures of *petit mal* or of *grand mal* type occur frequently in patients who may be assigned to any one of these groups.

I. *Infantile Hemiplegia.*—The *symptoms* are the sudden development of a unilateral paralysis after a series of convulsions attended by high fever and its attendant discomforts, and a period of unconsciousness of varying duration; then a gradual improvement in the paralysis after the active manifestations of the onset have subsided; and finally, a stationary condition, in which the face is but slightly affected in its voluntary or automatic movements; the speech is usually regained, if it had been lost; the arm is quite seriously paralyzed, the fingers being stiff and awkward, and sometimes being in constant slow involuntary motion (athetosis); the leg is held rather rigid, so that the child limps in walking, and often develops a club-foot. There are, of course, on the one hand, light cases in which the symptoms finally amount merely to clumsiness in the fingers. There are, on the other hand, severe cases in which a double hemiplegia has occurred, both halves of the body being equally affected, and in which both arms are useless and both legs so stiff, so closely adducted, and so helpless, that walking is impossible (diplegia). The latter are cases in which all the symptoms date from birth. In all cases the paralyzed limbs are found to be affected in their growth and development, so that they are smaller, colder, stiffer, and weaker than the others; the reflexes are exaggerated, but the electrical reactions are not qualitatively changed, and the sensation is normal. This condition remains through life as a permanent defect, and although the division of contractured muscles or tendons and the application of ingenious apparatus may correct deformities and make the paralyzed parts fairly useful, and although the application of electricity to the muscles chiefly affected may increase their nutrition and thus prevent contracture which comes from the unbalanced strain between the various muscles, yet any great degree of improvement is

impossible. In more than one-half of these cases epileptic attacks are of frequent occurrence.

The cases which date from birth must be divided into those in which there has been evidence of traumatism during labor, and those in which there was none. In the former class it may be stated confidently that cerebral hemorrhage, usually meningeal, is the cause of the symptoms. In the latter class it is probable that an intra-uterine encephalitis or some unknown cause has prevented the fœtal brain from developing.

The cases which have developed suddenly after birth are cases in which the ordinary causes of hemiplegia in adults have been acting, and must be traced either to encephalitis, hemorrhage, or embolism, or disease of the vessels. The degree of spontaneous recovery in any case can be pretty well determined by an examination at the end of the second year. A complete cure never occurs.

The fits in organic epilepsy are likely to be more frequent and severe than in idiopathic epilepsy; as many as twenty convulsions may occur daily for weeks at a time. These fits do not destroy life, but render life a burden. When, however, they are infrequent they may be benefited to some degree by the use of bromides.

II. The second class of cases presents mental defects more noticeable than physical symptoms. The child may be slow in learning to talk, may seem unable to fix its attention upon anything continuously, may be exceedingly active, in constant motion—the activity being, however, aimless; may throw things about, or tear things up, or put everything into its mouth; may be very difficult to manage because of its inability to retain and combine impressions with sufficient power to reason upon them; and may, therefore, be incapable of appreciating the meaning of punishment, if this be inflicted. Such children may have good powers of perception, may recognize persons and objects, show pleasure at bright colors, or music, or caresses, but fail to show evidence of thought in the sense of reasoning power, judgment, or self-control. Some patients constantly drule at the mouth, cannot be taught cleanly habits, and are manifestly imbecile. Other patients are quite bright in many directions, may even be precocious, show talents in music, or drawing, or fondness for mathematics, designing, languages; yet are apparently unable to appreciate moral ideas, cannot be taught to tell the truth, are cruel and bad, will not control any of their impulses, and so are the distress and despair of parents and teachers. It is those mental

qualities which are the product of the highest evolution which have failed to develop in this class of cases. The final result is that they have to be taken care of all their lives, either at home or by attendants, being incapable of supporting themselves or directing their conduct. Many of them have epilepsy. Some of them have hemiplegia.

III. The third class of cases is less common than the two preceding, and is likely to escape observation unless carefully investigated. The patients belonging to this class may be hemiplegic or may present no motor or mental defects, though they may be the subjects of epilepsy. They do have defects of sensory perception. Such defect may be in the form of hemianopsia or of deafness. It is probable that many cases of deafmutism belong to this class, and in some persons, as in Laura Bridgeman, deafness, dumbness, and blindness coincide.

The pathological changes in the brain in these various cases differ widely, and are as follows, in the order of frequency:

Porencephalus, a localized atrophy or agenesis, leaving a cavity in the cerebral hemisphere, which may be deep enough to open into the ventricle.

Sclerotic atrophy, an atrophic condition of the brain with an increase of connective tissue and disappearance of the nervous elements; affecting both hemispheres, or one only, or a part of one only; or limited to small areas in various parts.

Maldevelopment and apparent atrophic condition of the minute structures of the hemisphere, chiefly cortical, the cells resembling those of a new-born child, but with no apparent gross defects in the brain.

Atrophy, consequent upon the condition of softening produced by embolism or thrombosis, and limited in extent to certain arterial districts of the brain.

Meningo-encephalitis, a condition shown by thickening and adhesion between the pia and the brain, with destruction of the cerebral cells and atrophy of the cortex.

Cysts lying on the brain and producing atrophy by pressure, or associated with atrophy due to the original lesion of which the cyst remains as a trace.

Hemorrhage on or in the brain, as shown by the remains of a clot, or by hæmatin staining of a cyst, of the pia, or of the sclerotic tissue.

Hydrocephalus with extreme dilatation of the ventricles, so that the brain-tissue is reduced to a mere wall about the cavity.

These are the conditions found at death in cases presenting the clinical features just studied. It is evident that they have this in common, namely, a condition of atrophy of the brain. The origin of this atrophy is not always clear. In some cases it is clearly congenital and due to a maldevelopment of the embryo. In other cases it is clearly traceable to injuries at birth. In other cases it must be ascribed to affections of various kinds, such as inflammations of the membranes or of the brain substance, or vascular lesions and their consequences, such as occur in adults. But it is evident that the various processes of disease have as a fairly uniform result a condition of atrophy with sclerosis of the brain, which may be termed sclerotic atrophy. The difference in the clinical types is due to the varying situation of the lesion rather than to its varying nature.

For such conditions there can be no treatment.

Tumors of the Brain.

Varieties in order of frequency:

Gumma: soft gelatinous, or hard cellular, situated on base or in cortex, usually beginning in meninges.

Tubercular: hard masses, often multiple, especially frequent in children, situated anywhere, sometimes primary, usually secondary. This is the variety in more than one-half the cases in children.

Glioma: peculiar to the nervous system, cells and fibres like the neuroglia, usually infiltrated and without boundaries, but single, usually vascular; hence symptoms vary and hemorrhage occurs; usually situated in the white substance.

Sarcoma of all varieties: fibro-, round-cell, spindle-cell, glio-sarcoma, begin from connective tissue of membranes as a rule, sometimes in the brain. Hard single tumors, of various sizes, easily separable and well defined, situated on base, in meninges, in cerebellum.

Carcinoma: rare, usually secondary.

Occasional varieties are fibromata, osteomata, psammomata, cholesteatomata, angiomata, lipomata, ecchinococcus, and cysticercus.

Aneurisms of the base give rise to symptoms of tumor.

For Pathology of Tumors, see Delafield and Prudden's "Pathology."

Causes of Brain Tumor.—1. Predisposing: Sex: males twice as

In aneurysm of the brain there may be a bruit over the temporal region.

frequently affected as females. Age: children most liable, as tubercular tumors are frequent. Young adults next. Old age is quite exempt, except from carcinoma.

2. Exciting. Injuries. Severe emotional shock. Unknown in large majority.

Situation.—Cerebral cortex, twenty-five per cent.; cerebellum, twenty-five per cent.; centrum ovale, fifteen per cent.; basal ganglia, ten per cent.; pons, ten per cent.; crus and corpora quadrigemina, ten per cent. Others, five per cent.

Effects of Brain Tumor.—1. Direct effects. Irritation of gray matter with nervous discharges, and destruction of brain tissue.

2. Indirect effects. General pressure on entire brain. This may be limited by the falx or tentorium. It produces hydrocephalus by pressure on ventricle or veins of Galen. Vascular disturbance by pressure on arteries and veins. Meningitis, localized and not purulent, except in tubercular cases. Thinning of cranial bones.

Effects of increased pressure are seen after death in flattening of the convolutions, anæmia and pallor of the brain, dry sticky state of membranes, and distention of ventricles by serum.

Symptoms of Brain Tumor.—I. General symptoms depend on existence of a new growth independent of its position.

Headache in ninety-five per cent., constant or intermittent; general or local; dull and deep, increased by anything which deranges the cerebral circulation. Sometimes head is tender to percussion at certain points; may have evidence of pain even in state of stupor; it may be agonizing; it may give feeling of band about head; it is often worse at night, causing insomnia, especially in gumma.

Mental disturbance, eighty-five per cent. Dulness and apathy, with imperfect attention and memory. Depression and emotional state. Slow speech, gradual mental failure, childishness, and dementia; fainting attacks, stupor, and coma in the last stage. Occasionally delirium and active insanity.

Optic neuritis, eighty per cent. Venous congestion. Swelling of papilla, loss of disc outline; radiating appearance about the disc and hemorrhages; then optic-nerve atrophy leaving white or gray disc. In cases of subcortical tumor of small size it may be absent. Sight may be affected; visual field small and irregular, or unimpaired for a time.

Vertigo or sense of giddiness, frequent in tumors in posterior

fossa. May occur in tumors anywhere on change of position. May be due to paralysis of one or more ocular muscles.

Vomiting, with or without nausea or relation to food. Frequent in tumors in posterior fossa.

General convulsions of epileptic type. *Petit mal* or *grand mal.*

Nystagmus, lateral oscillation of eyes. Frequent in disease of corpora quadrigemina and cerebellum.

Loss of control over sphincters occurs, especially with tumors in the frontal lobes.

Glycosuria and polyuria, especially with tumors in the posterior fossa.

Slow pulse; 50 to 60.

II. Local symptoms dependent upon the position of the tumor.

Frontal Lobes: Mental dulness, lack of control of emotions, irritability, inattention, and childishness, dementia, loss of sense of smell.

Left third frontal convolution. Motor aphasia.

Central Convolutions, and motor tract. Monospasm and monoplegia when in or near cortex. Hemispasm and hemiplegia when deep or in capsule. Sometimes slight anæsthesia in paralyzed part.

Occipital Lobe and visual tract. Hemianopsia, noticed by patient as unilateral blindness. Visual hallucinations and flashes of light on one side.

Left Temporal first and second convolutions. Sensory aphasia: form, word-deafness.

Left Lower Parietal lobule and angular gyrus or in occipito-temporal tract. Sensory aphasia: form, word-blindness.

Base of brain.

Anterior fossa: Loss of smell.

Middle fossa: Hemianopsia, paralysis of ocular muscles, III., IV., VI.; neuralgia of face and anæsthesia, V.; paralysis of III. and opposite hemiplegia, neuroparalytic ophthalmia, nystagmus; bilateral spastic paralysis, deafness.

Posterior fossa: Paralysis of VII., IX., X., XI., XII.; vertigo and aural symptoms; cerebellar ataxia, tendency to one side; paralysis widespread from medulla disease.

Cerebellum: Vertigo, vomiting, cerebellar ataxia when middle lobe is involved; headache, frontal or occipital and severe; optic neuritis early.

Diagnosis.— Cerebral symptoms: general alone, or general + local, point to the presence of a tumor.

Differentiation from chronic nephritis: By examination of urine; by condition of heart, arteries, and blood-pressure; by appearance of optic disc and retina in many cases, hemorrhages more frequent and white spots far from disc; by character of headache, more severe in tumor; by age of patient, young person = tumor.

From hypermetropia with astigmatism and anæmia, with menstrual disorder: By examination of eyes and relief of glasses; by cure of anæmia by diet, iron, and aloes; by slight degree of headache and neuritis.

From hysteria with migraine: By study of the patient and separation of real symptoms; by condition of the optic disc.

From abscess: By history of trauma or of otitis media; by severity of early symptoms in abscess; by fever in abscess; by late development of optic neuritis, rare in abscess.

To determine the location: Study carefully the local symptoms, "signal symptom," *e.g.*, numbness and twitching of thumb. Study also the order of appearance of local symptoms: thumb, hand, face. Compare the several local symptoms, paralysis + aphasia, or + hemianopsia.

To determine the variety: Estimate probable frequency of various tumors at different ages. Look for history of syphilis, tuberculosis, cancer, or sarcoma. Study rapidity of onset; glioma, tubercle, cancer are rapid. Study signs of irritation; more frequent in glioma and meningeal tumors. Study signs of variation in severity of symptoms; more variation in glioma. Study situation of the tumor; basal tumors are often gumma, tubercle sarcoma. Intracerebral tumors are often glioma, sarcoma. Peculiar forms of hemiopia and early optic neuritis indicate sarcoma of pituitary body or aneurism.

Course of the disease: Begins with general symptoms usually. Sometimes begins with local spasm or a general convulsion. Gradual increase in severity and number of symptoms. Finally death in stupor or in general convulsion or suddenly, from heart failure, or recovery under specific treatment.

Duration is from one to three years.

Prognosis bad, excepting in gumma and when tumor can be removed.

Treatment.—For gumma: Inunctions of mercury and KI to three hundred grains daily.

Surgical treatment: Removal is possible when diagnosis of location is exact. There is danger of return.

For Cranio-Cerebral Topography, see page 44.

Abscess in the Brain.

Acute encephalitis with formation of pus results in an abscess. The abscess may extend rapidly, not being encapsulated, and cause death; or it may become encapsulated and develop slowly, or become stationary and remain for years as a foreign substance producing no symptoms, but finally enlarge or break. Occasionally multiple abscesses develop in the brain secondary to pyæmia, producing cerebral symptoms in the course of that disease. There may be a secondary meningitis in either form.

Situation.—Is usually in the temporo-sphenoid lobe or in the frontal lobe or in the cerebellum, in the white matter.

Causes.—Chronic otitis media, forty per cent. Fracture of cranial bones, thirty-five per cent. Chronic rhinitis, ten per cent. Caries of any cranial bone, ten per cent. Secondary to infectious diseases, five per cent.

Symptoms.—After an injury or after otorrhœa or other cause cerebral symptoms develop suddenly; severe headache, vomiting, mental distress and dulness, stupor, a chill, a high temperature for a day or two; possibly convulsions; rarely local symptoms; hemiplegia, hemianopsia, aphasia, cerebellar staggering. In a week the patient is very ill, with irregular temperature, sometimes with chills, sometimes no fever; slow pulse; intense pain, delirium, stupor, and optic neuritis. If abscess extends death follows in two weeks, or suddenly from rupture. If abscess discharges into ear or nose, or if it can be opened, symptoms subside. Rarely symptoms subside spontaneously slowly, the abscess becoming encapsulated and latent, and patient apparently recovers, but is subject to headache and vertigo; and has a relapse later.

Diagnosis.—

Abscess	*vs.*	Meningitis.
Temperature normal or not high.		Temperature high without remission.
Pulse slow.		Pulse rapid.
Patient in stupor.		Patient irritable, all senses overacute.

Anything increasing intra-cranial pressure causes a slow pulse.

Causes:- Sequel of infectious diseases.

Spasms and rigidity rare.	Spasms and rigidity frequent.
Cranial nerves not involved.	Cranial nerves involved early.
Optic neuritis frequent.	Optic neuritis rare.

ABSCESS	*vs.* THROMBOSIS OF LATERAL SINUS.
Temperature normal or not high.	Temperature high with remissions.
Pulse slow.	Pulse rapid, weak.
Chill at onset not repeated.	Chills frequently repeated.
Little sweating.	Profuse sweating.
Head tender to percussion?	Pain over mastoid and jugular.
	Thrombi in lungs.

Diagnosis of location is made partly from the exciting cause and partly from the local symptoms.

Prognosis fatal, unless abscess can be opened.

Treatment.—In the few cases in which the abscess can be located, operation of trephining and drainage.

CEREBRO-SPINAL SCLEROSIS.

Multiple or disseminated sclerosis is a rare disease affecting the brain and spinal cord together. Small regions of sclerotic tissue are found irregularly scattered through the entire nervous system, without destruction of the axis cylinders of the nerve-tracts. Its cause is unknown.

Symptoms.—Headache, vertigo, malaise, mental irritability, inattention, imperfect memory, lack of self-control, and inability to work. Later, tremor of hands increased by an effort to hold them still, nystagmus, increased knee-jerk, stiff spastic gait, weakness, and finally general tremor on effort. Slow speech, each syllable separately enunciated without variation of tone. Optic atrophy. Dementia and attacks of epilepsy and hemiplegia may finally occur.

Course is exceedingly irregular, symptoms may disappear for months and then return. The disease is often mistaken for hysteria.

Diagnosis is made by excluding all other organic diseases, by nystagmus, tremor, and speech.

Treatment.—Physostigmine, $\frac{1}{60}$ gr. daily; solanin, $\frac{1}{2}$ gr. daily, increased.

Sml. constant doses of As.—Acid As. 1/50 gr.
alternating w/ Bichloride 1/50 gr. t.i.d.

Syphilis of the Brain.

Syphilitic affections of the nervous system may be produced by:

1st. Syphilitic endarteritis.

2d. Direct action of the syphilitic poison upon the nervous system.

3d. Syphilitic exudations in the meninges.

4th. Syphilitic deposits in the brain itself.

5th. Hereditary syphilis.

1st. Syphilitic endarteritis produces a progressive diminution in the calibre of the blood-vessels, resulting in a state of anæmia, malnutrition of the brain supplied by the vessels affected, and leads to thrombosis followed by localized softening in the area cut off from its blood supply. The symptoms of this condition are in the early stage those of malnutrition of the brain and imperfect action in the regions affected. The patients present at first symptoms of neurasthenia, and later slight temporary suspension of functions, such as temporary aphasia, numbness in one limb or in one-half of the body, a condition of weakness not amounting to paralysis in one limb or one-half of the body, double vision, or vertigo. Stimulants to the heart relieve these symptoms, but after a time if a thrombosis occurs the symptoms of cerebral apoplexy suddenly appear. After such an apoplectic attack a partial recovery may ensue, but as a spot of softening in the brain is usually left, a complete recovery is not to be expected; hence the fact that a syphilitic lesion which can be removed by treatment is the cause of the apoplectic attack does not warrant a more favorable prognosis in such cases.

2d. The toxic effects of syphilis on the nervous system are manifested by general disturbances of function, which produce the symptoms of neurasthenia of all the various types. As a rule, in syphilitic neurasthenia the symptoms appear to be worse toward evening and insomnia is more persistent than any other symptom; hence in general neurasthenia with insomnia any syphilitic element must be looked for and treated before the neurasthenia can be cured. Neurasthenia may occur in syphilitic persons without any direct connection with the syphilis, which at the time may be latent. A test of treatment is the only one enabling a differential diagnosis to be made.

3d. Syphilitic exudations into the meninges of the brain and

In 90% of neurasthenics it is bad t
give anti-syph. treat.
In syph. neurasthenia the symptoms
are worse late in the afternoon. Rest
less insomnia indicates syph. orig

syphilitic meningitis are exceedingly common as a sequel of syphilitic disease. From two to ten years after the infection gummy exudations may occur in any part of the brain, but are more frequent upon the base of the brain and about the crus and pons. The glue-like substance is deposited rapidly and extensively through the meninges, producing pressure upon the subjacent brain or upon the cranial nerves, and thus causing suspension of function in the parts compressed. Occasionally the syphilitic exudation takes the form of a hard tumor, which then produces the regular symptoms of tumor of the brain. The symptoms of syphilitic gumma or syphilitic basilar meningitis are headache, worse at night, insomnia, mental irritability, vertigo, distressing sensations in the head, occasional vomiting, and local symptoms of paralysis of the cranial nerves, the third and sixth being the ones particularly affected. If the exudation is not upon the base, or occurs in the convexity or over the cerebellum, the local symptoms will be those of irritation or of arrest of function of the particular portion of the brain affected. Thus spasms and paralysis, paræsthesia and anæsthesia, hemiopia, or any variety of aphasia may be caused by the disease. The pressure upon the vessels produced by syphilitic exudations adds to the complexity of the symptoms. Convulsions are frequently observed in such cases, both of a general and of a local type. Increased thirst and hunger and polyuria are often found. The course of the case is more rapid in its onset than that of brain tumor, but resembles it in all other respects and may be associated with optic neuritis. The symptoms subside rapidly under inunctions of mercury and large doses of iodide of potash, and hence prognosis is good in the majority of cases, though relapses are frequent.

4th. Syphilitic deposits in the brain itself may be of the nature of small disseminated spots, producing chronic indurative or sclerotic processes or small regions of softening. The symptoms of this affection are identical with those of general paresis, and can only be distinguished from paresis by the result of treatment.

5th. Hereditary syphilis occasionally produces the symptoms of cerebral atrophy or the symptoms of multiple cerebro-spinal sclerosis in children, and in either of these conditions this fact must be kept in mind, and if other signs of hereditary syphilis are discovered this element must be considered in the treatment.

FUNCTIONAL NERVOUS DISEASES.

Neurasthenia.

Nervous prostration, nerve tire, nervous irritable weakness is a functional disease of the entire nervous system, brain, spinal cord, spinal and sympathetic nerves, dependent upon malnutrition, characterized by nervous weakness and irritability.

A balance between expenditure of energy and nutrition must be preserved.

Brain overwork or anxiety;

Spinal cord overwork by long marches or sexual overaction;

Nerve overwork by various occupations in which a single act is repeated;

Vasomotor work in maintaining vascular tone and nutritive processes, all require a sound basis and rest at periods.

When balance between store and expenditure of energy is broken, exhaustion results. Then nature enforces rest by fatigue, by sleep. One may disregard nature, but if so one must pay the penalty.

Causes.—I. Excessive expenditure of nerve energy.

Primary neurasthenia more common than secondary.

Influences favoring its production:

1. Bad hereditary influences. Weak nervous system.
2. Feebleness in childhood, with poor nervous system.
3. Wrong methods of training and education.
4. The struggle for existence. Kant's rule, eight hours for work, eight hours for diversion, eight hours for sleep.
5. Anxiety, worry, mental depression, fear.
6. Mental or physical overwork.
7. Sexual excesses.

II. Deficient supply of nervous energy.

Secondary neurasthenia.

Influences favoring its production.

1. Weakening diseases of all kinds of organic nature.
2. Indigestion and dyspepsia, with auto-infection by products.
3. Gout, rheumatism, uric-acid diathesis.

4. Infectious diseases, typhoid, grippe, malaria.

5. Alcoholism or abuse of drugs.

Result of the action of these causes is to produce malnutrition of the nervous system and state of irritable weakness.

1. The power of continuous activity is impaired; weakness results; hence, mental work is imperfect; attention flags; memory fails; ambition ceases; physical activity is imperfect; exercise tires one; lassitude; vegetative functions are impaired. Digestion is poor; constipation occurs; vascular tone is impaired; heart feeble. Balance must be maintained between blood supply and functional activity. When this is impaired work is impossible. Secretions are changed in amount and character, *e.g.*, urine.

2. The power of mental and physical control is impaired and irritability results; inhibition may be partly automatic and partly conscious; it must be conscious and voluntary to prevent morbid fear and apprehension; influence of fear is to inhibit all nervous acts—*e.g.*, walking on ice, oral examination, stage fright; this control is weakened, and hence irritability results.

Symptoms of Neurasthenia.—

1. Cerebral: Headache—dull, occipital, frontal; sensitiveness of scalp; vertigo; insomnia; cerebral sensations—fulness, band, pulsation.

2. Mental: Apprehension—morbid fears, of places, people, solitude, etc.; inattention and forgetfulness; incapacity for work; idiosyncrasies become prominent; irritability of temper ensues; anxiety and exhaustion; imperfect respiration and rapid pulse.

3. Spinal: Pain, especially in nape of neck and sacrum; irritation; hypersensitiveness; sensitiveness about ribs and intercostal nerves; weakness, legs give out; sexual irritation, erections, impaired power; emissions; bladder irritability, frequent micturition.

4. Vasomotor: Flushes, cold extremities; undue sweating; transient blueness or œdema; *tache cérébrale* of Trousseau; tachycardia; palpitation.

5. Gastro-intestinal: Indigestion and dyspepsia, especially acid stomach; imperfect liver action; constipation; pseudomembranous colitis; distention of abdomen with gas.

6. Sensory and motor symptoms: Indefinite pains and paræsthesia; joint affections; apparent paralysis—no change in electric activity; vision imperfect; asthenopia; visual field may be retracted. Other senses rarely affected; tinnitus.

Stay in bed until 10 or 11 A.M. - let him have coffee 8 A.M. - 8.15 wash face & teeth 8.30 breakfast - rest an hour - 10 to 10.30 let him have paper or letters - 10.30 give him a pack for 20 min. now let him sit or stand up & sponge him off w/ H_2O at 80 gradually reduced to 70, if weak circulation rub off w/ towel dipped in brine. Now let him dress - then rest - until 12.30. Now let him sit at open window or take a drive - no walk. At 1 let him have good meal, rest from 1.30 to 2.30 - now read for 1/2 hour sitting in chair. 3 P.M. let them go out again for fresh air. 4 P.M. return - lie down & undress - massage for 1 hour - rest for 1 hour - giving little drink - rest for 1 hour - 6 P.M. get up at 7 dinner - lie down for 1/2 hour - read for 1/2 hour - 9 P.M. sponge bath or hot bath for insomnia - then go to bed

Medicines: - Tonics - bitters before meals - 1 - 2 tabs after meals.
Glycerophos. soda - phosphorous - iron -
Follow food by HCl or alkalies if too much acidity.
If insomnia is very marked - sedatives may give frequent sm. doses as 5 gr. Na Br every 2 hrs.

Diagnosis made by the existence of a cause; the absence of objective signs of organic disease; the temperament of the patient; the variability of symptoms; the disproportion between complaint and actual power.

Treatment.—Absolute necessity of establishing nerve-energy by restoring balance between store and expenditure. Hence supply nutritive elements to nervous system by proper food, properly digested; by increasing circulatory power; by exercise; by hydrotherapy; by massage; and diminish expenditure of nervous energy by rest, if necessary absolute, and by diversion.

Direct treatment may be by tonics of all kinds; by sedatives, if patient is extremely nervous; by electricity; galvanism. Treat symptoms only when absolutely necessary. Combat the desire of these patients for drugs, and their dependence on stimulants and opiates.

Hysteria.

A functional disturbance of the nervous system characterized by mental and moral perversion, lack of self-control, and disorders of any or all of the bodily functions.

Women, between fifteen and twenty-five years, or at any age.

Men, at any age, and even children, may be affected.

Causes.—Hereditary influence very marked; malnutrition of the nervous system from anæmia; indigestion; from bad habits of life; emotional excitement; fear, anxiety, jealousy; tendency to imitation yielded to; epidemics of hysteria; uterine and ovarian irritation is a rare cause. Traumatism, especially accidents attended by great fear. Several causes may concur.

Symptoms.—1. Mental changes, and moral perversion.

Increased irritability, and emotional excitability with impaired self-control.

Excessive emotion leads to exhaustion.

Craving for notice and sympathy.

All sorts of means taken. Self-injury; sores which won't heal; needles swallowed; blood sucked and vomited; animals concealed.

Purposeless acts of criminal kind, like reasoning mania.

Hallucinations may occur.

Depression is not uncommon, and melancholia may be feared. But in melancholia facial expression is characteristic, thought is slow, delusions are of self-accusation, weight is rapidly lost.

2. Disorders of various functions:

(1) *Motor.*—Spasms, contractures, paralysis, convulsions, globus hystericus, vomiting, diarrhœa, vesical irritation, convulsion attended by sobbing and crying, lasts longer than an epileptic fit, does not begin or end suddenly, and tongue is not bitten, and there is no asphyxia. Paralysis, varies in intensity, does not affect the face in hemiplegia, no typical gait, no bedsore, no incontinence; aphonia, aphasia, cough.

(2) *Sensory.*—Hyperæsthesia of special senses, dislike of light or noises; hyperæsthesia of skin, spinal irritation; hyperæsthesia of limbs, numbness; hyperæsthesia of ribs, pressure of dress; hyperæsthesia of ovaries; hyperæsthesia of bladder, frequent micturition; pain anywhere, rarely definitely localized; especially in joints, hip, knee, ankle, wrist, elbow; fixation, atrophy of muscles; joints easily moved under ether; no deformity; anæsthesia to pain, usually localized, in skin or mucous membranes; unilateral loss of special senses.

(3) *Vasomotor.*—Palpitation and faints; variations of arterial tone, flashes, œdema; hæmatemesis, hæmoptysis; small hemorrhagic spots, stigmata; rises of temperature, 105° to 110° F.

(4) *Secretion.*—Saliva; gastric juice varies. Urine — or + .

Hysterical attacks: Crying and laughing, produced by emotional strain; scream, convulsion, opisthotonus, facial expression, series of short attacks, fit of crying, passes water, cataleptic rigidity, hystero-epilepsy.

The character of the symptoms varies greatly. The symptoms appear and disappear suddenly.

The course of the disease: Chronic condition with periods of intensity; cure, sudden and marvellous; great variation in symptoms; very rarely death in convulsion or from suicide.

Diagnosis is never made from a single symptom, but from a study of the general condition.

Treatment.—Mental change and moral education; hydrotherapy; the rest cure; mental therapeutics; tonics, strychnine, iron, and quinine.

Symptomatic remedies:

Sedatives for the nervous state, bromides, valerian, lavender, asafœtida. Opium is to be avoided, for habit is easily formed.

For convulsion, prevent injury, don't use chloroform, use cold water, hold patient, stop respiration, don't press ovaries, give emetic.

water or abdomen, surround w/- dry towel - fresh air & a little exercise before going to bed - making deep respiratory efforts.
Drugs to produce sleep act best when given w/ a little hot food before going to bed.
For vertigo - KBr or strontium bromide sm. frequent doses. 5 grs. every 1/2 hour
For headache - combine several coal-tar derivatives w/- a sm. amt. of a cardiac stimulant.

Phenacetine	6 grs
Acetanilid	3 "
Caffein	1/2 "
w/- or w/out Sod. Bicarb.	5 "

Sometimes strych, ignatia, or brucia sm. doses relieve headache -

1/200 or 1/250 strych
or 1/100 — — ignatia
or 1/200 — brucine

every 10 min. for 4 or 6 doses
Don't let pt. have opium or morphine codeine or celerina. In extreme cases may give a 1/30 gr. of codeine every hour for 5 doses as for sending a lawyer over a c
Stimulants: — Should have no alcoholic stimulants.

Hysterical paralysis differs from true paralysis in that the paralysis in hysteria is complete tho effort to move the limb is made.

For faints, irritation over heart by ice, internally spirits ammonia, or lavender.

For spasms, hot or cold applications, cautery.

For paralysis, electricity, especially faradism.

For pain, hypodermics of water after one of morphine, local applications of sedatives.

IG. 1.—Normal Nerve Fibre. *a*, Axis cylinder; *b*, medullary sheath; *c*, sheath of Schwann; *d*, node of Ranvier; *e*, incisure of Schmidt; *f*, nucleus of sheath of Schwann.

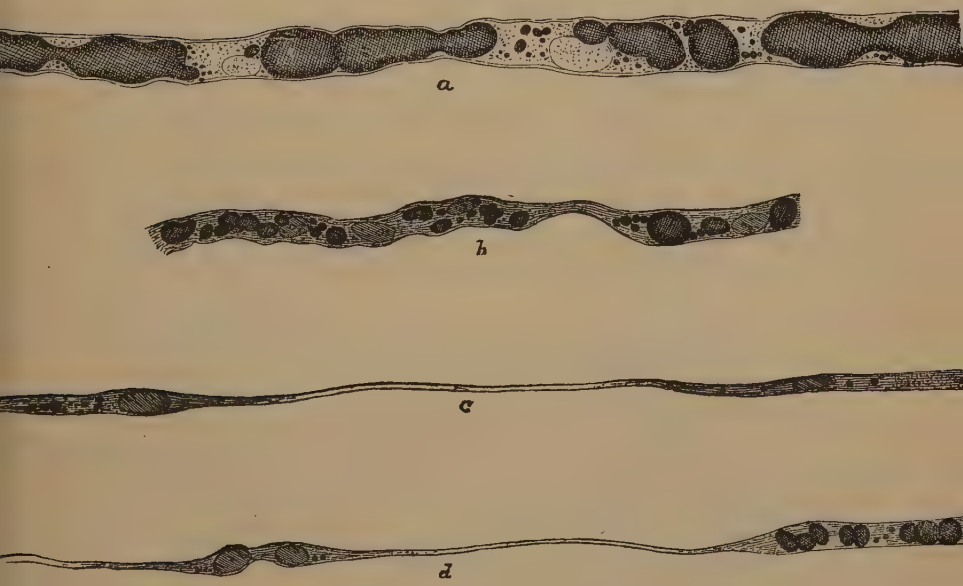


FIG. 2.—Degeneration of Nerve Fibres in Various Stages.

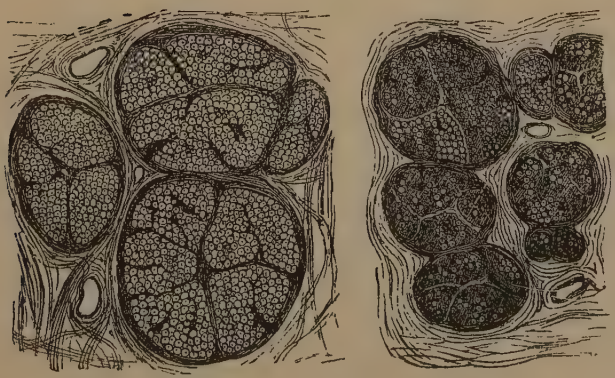


FIG. 3.—Cross-Section of Normal and Degenerated Nerve Trunk.

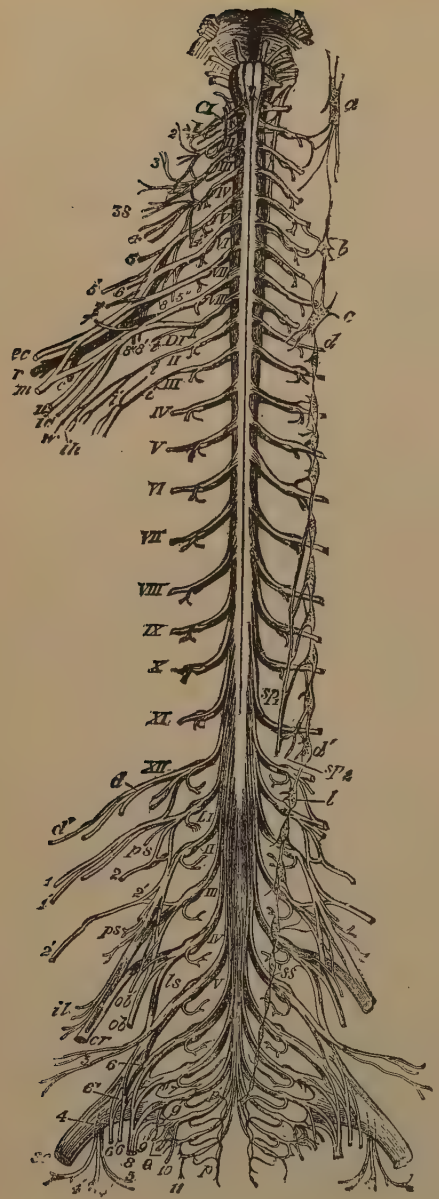


FIG. 4.—The Spinal Cord with its Nerves (Anterior Surface). Brachial, Lumbar, and Sacral Plexuses and Nerves of Extremities shown on left; Sympathetic Ganglia with Spinal Roots shown on right of the figure.

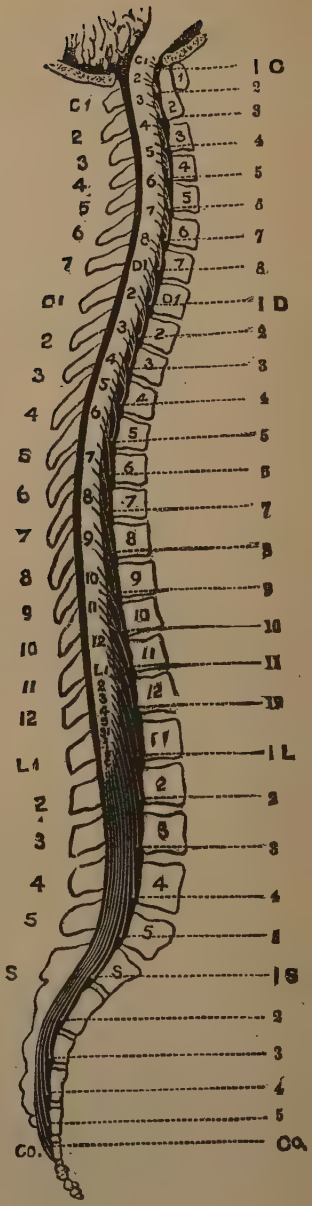


FIG. 5.—The Relation of the Spinal C to the Vertebral Bodies and Spines. The ends at *L I*.

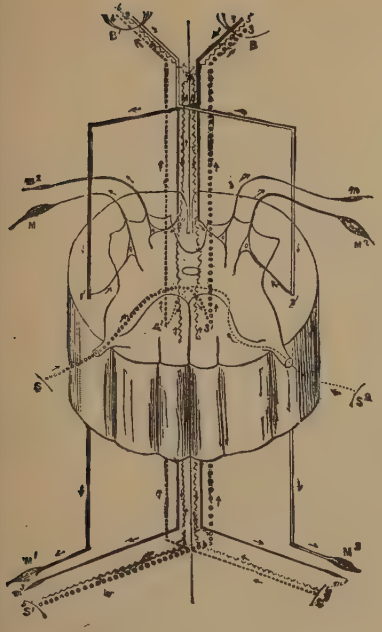


FIG. 6.—Diagram of a Spinal Segment as a Spinal Centre and as a Conducting Medium (Bramwell). *B*, Right ; *B'*, left hemisphere ; *MO*, medulla oblongata ; 1, motor tract from right hemisphere; at *MO* it divides into a direct and a crossed tract, the former passes down the right ant. median column, the latter passes down the left lateral column, giving off at 1 fibres to the ant. gray horns of the cord, in which the motor cells lie, from which nerves pass to muscles, *M* ; 2, motor tract from left hemisphere ; *S*, *S'*, sensitive areas on the left side of the body; 3, 3', the main sensory tract from the left side of the body; it passes into the post. gray matter of the segment, then decussates, and passes up the right half of the cord in the post. columns, thence to the right hemisphere of the brain; 4, 4', the main sensory tract from the right side of the body; 5, 6, the tracts of muscular sense from the legs, passing up upon the same side of the spinal cord in which it enters, in the column of Goll, decussating at the medulla above the motor decussation. The arrows indicate the direction of nerve currents.

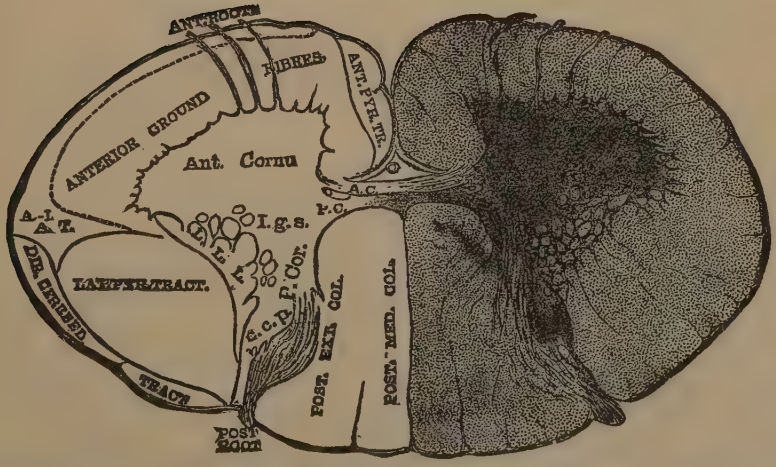


FIG. 7—Diagram of a Section of the Spinal Cord in the Cervical Region. *A.C.*, Ant. commissure; *P.C.*, post. commissure; *I.g.s.*, intermediate gray substance; *P.Cor.*, post. cornu; *c.c.p.*, caput cornu post.; *L.L.L.*, ateral limiting layer; *AL-AT.*, antero-lateral ascending tract.

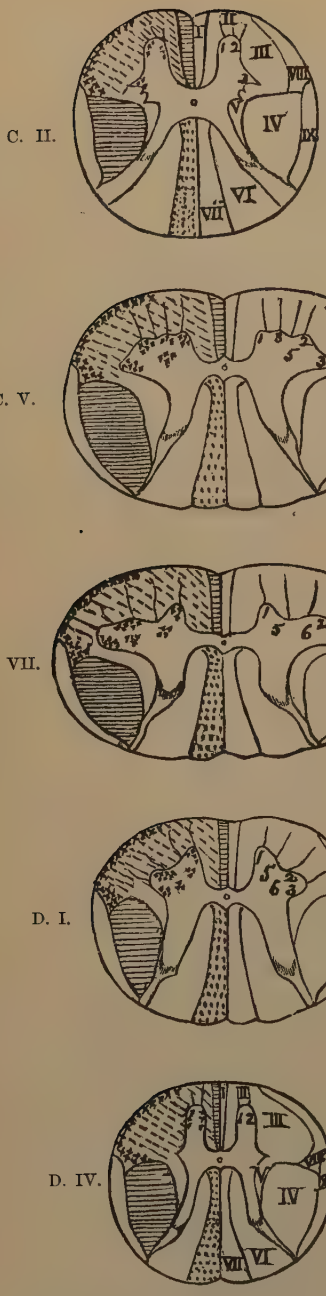


FIG. 8.—Sections of Spinal Cord. 1 to 6, Groups of cells in ant. horns; *I.–IX.*, columns of cord; *I.*, antero median; *II.*, anterior; *III.*, antero-lateral; *IV.*, lateral pyramidal; *V.*, lateral limiting layer; *VI.*, postero lateral or Burdach; *VII.*, postero-median or Goll; *VIII.*, antero-lateral ascending; *IX.*, direct cerebellar Between *IV.* and *VI.* column of Lissauer.

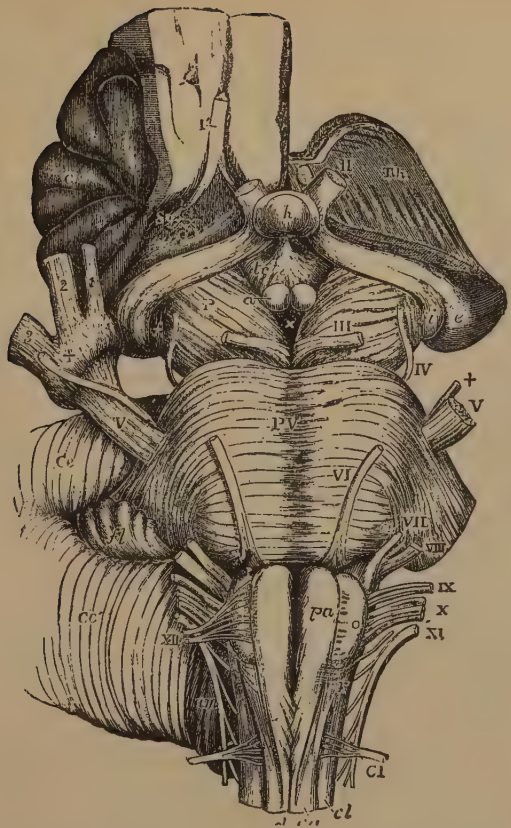


FIG. 9.—The Base of the Brain and the Cranial Nerves, Crura, Pons, and Medulla (Allen Thompson). *I* to *XII*, the cranial nerves; *Th*, optic thalamus; *h*, pituitary body; *tc*, tuber cinerium; *a*, corpora albicantia; *P*; pes pedunculi; *i*, interior, and *e*, exterior, geniculate body; *PV*, pons Varolii; *pa*, anterior pyramid of medulla, *o*, olive; *d*, decussation of anterior pyramids; *ca*, anterior column of spinal cord; *cl*, lateral column of spinal cord; *Ce*, cerebellum; *fl*, flocculus of cerebellum.

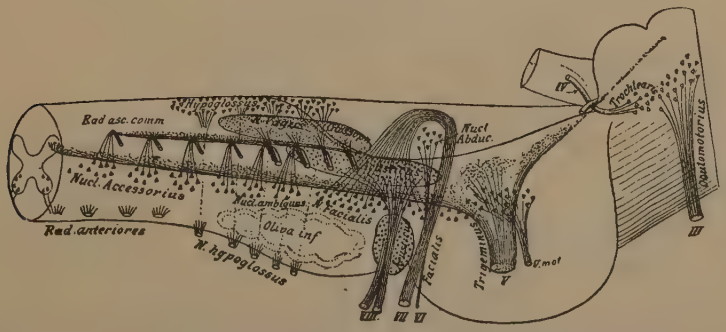


FIG. 10.—Longitudinal Section showing the Relative Position of the Cranial Nerve Nuclei (Edinger).

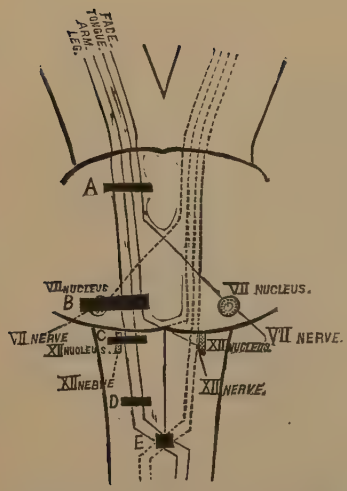


FIG. 11.—The Motor Tract in the Crus, Pons, and Medulla. *A*, Lesion causes hemiplegia. *B*, Lesion causes alternating paralysis; left facial, right extremities. *C*, Lesion causes paralysis of limbs and tongue. *D*, Lesion causes paralysis of limbs of opposite side. *E*, Lesion in motor decussation causes bilateral paralysis.

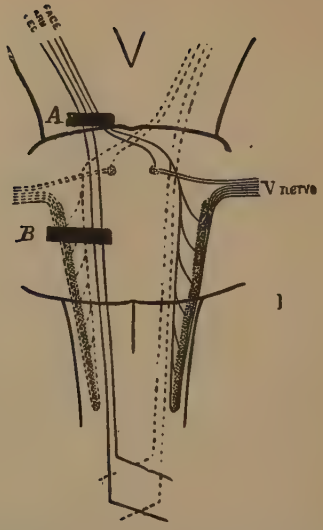


FIG. 12.—The Sensory Tract in the Crus, Pons, and Medulla, showing Nucleus and Roots of V. Nerve. *A*, Lesion causes hemianæsthesia. *B*, Lesion causes alternating hemianæsthesia; left face and right side of body.

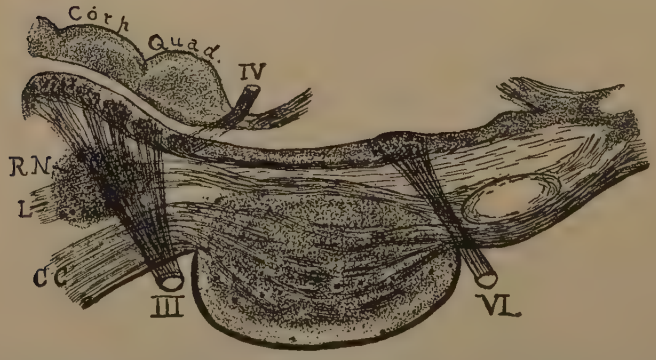


FIG. 13.—Sagittal Section through the Cerebral Axis to show the Nuclei of the Ocular Nerves in the Floor of the Aqueduct of Sylvius and Fourth Ventricle and the Course of the Nerves to their Exit. The various groups of cells from which the III. N. arises are seen. *RN*, Red nucleus of tegmentum; *L*, lemniscus (sensory tract); *CC*, motor tract in the crus cerebri seen to traverse the pons and enter the ant. pyramid of the medulla.

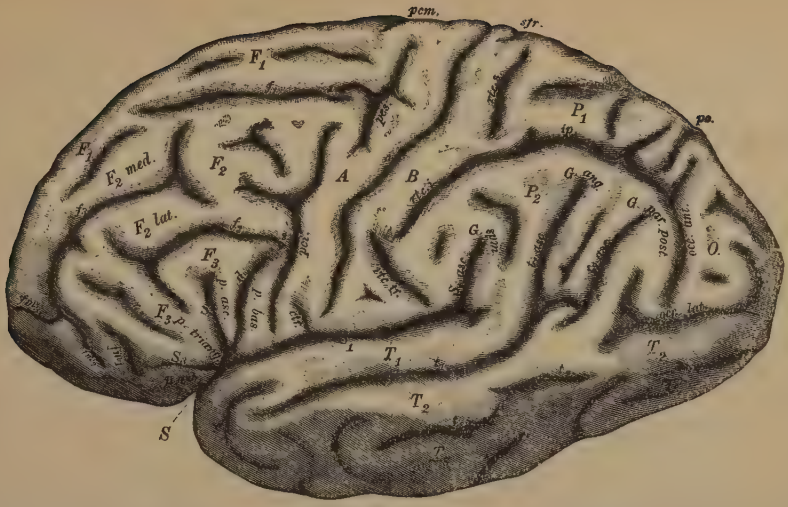


FIG. 14.—The Lateral Surface of the Left Hemisphere (Eberstaller). *S*, Fissure of Sylvius; *C*, fissure of Rolando, or central fissure; *pci*, sulcus præcentralis inferior; *pcs*, sulcus præcentralis superior; *rtc*, sulcus retrocentralis; *sfr*, sulcus subfrontalis; *f*1, *f*2, *f*3, sulcus frontalis, sup., inf., et medius; *ip*, interparietal fissure; *po*, parieto-occipital fissure; *occ. ant.*, *occ. lat.*, fissura occipitalis, ant. et lat.: *t*1, *t*5, *t*3, sulci temporales; *A*, *B*, ant. and post. central convolutions; *F*1, *F*2, *F*3, frontal convolutions, sup., med., inf.; *P*1, *P*2, parietal convolutions. sup. and inf.; *G. spm.* gyrus supramarginalis; *G. ang*, gyrus angularis; *O*, occipital lobe; *T*1, *T*2, *T*3, temporal convolutions.

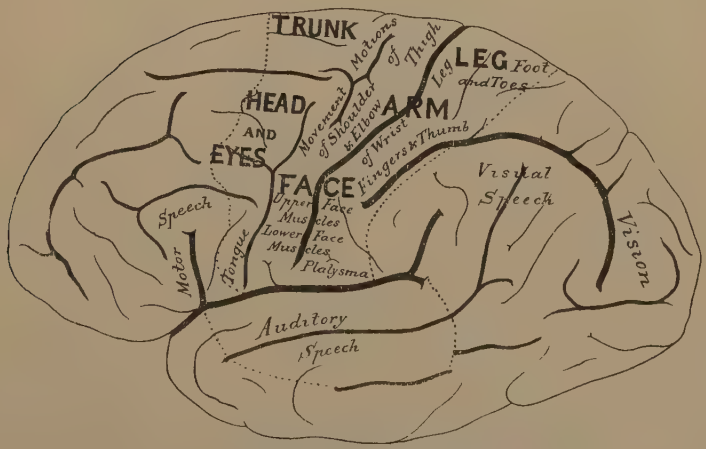


FIG. 15.—The Functional Areas of the Brain, Left Hemisphere.

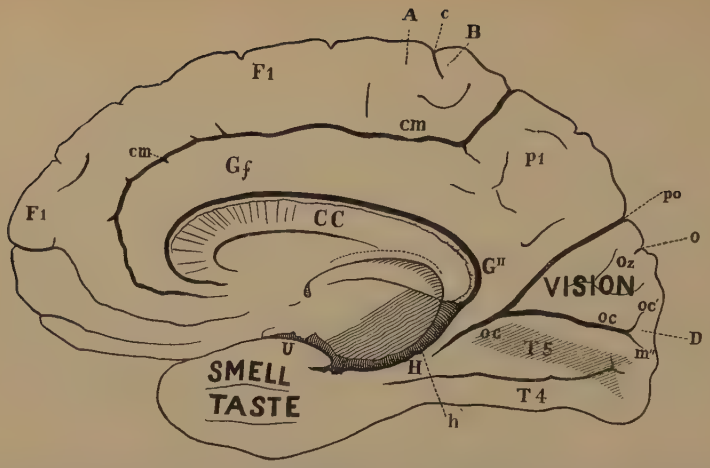


FIG. 16.—The Median Surface of the Right Hemisphere (after Ecker). The visual centre is in the cortex lying within the calcarine fissure, *OC*, and in the cuneus, *Oz*. The sensations of smell and taste are received in the uncinate convolution, *U*, and at the tip of the temporo-sphenoidal lobe. *A*, *B*, shows the position of the paracentral lobule, which is included in the motor area of the leg.

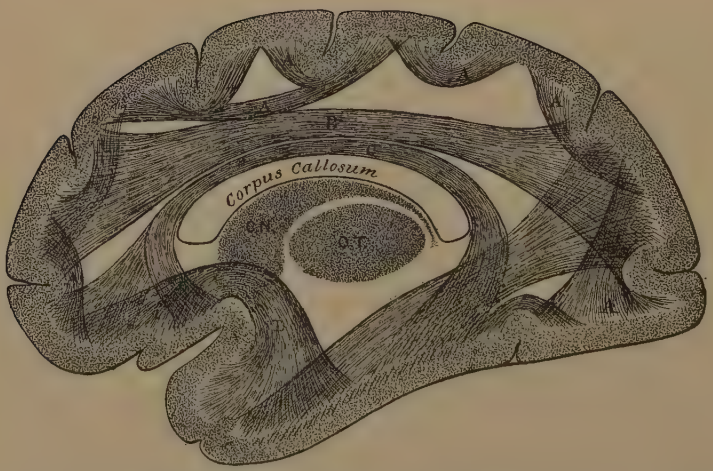


FIG. 17.—The Association Fibres. *A*, Between adjacent convolutions; *B*, between frontal and occipital areas; *C*, between frontal and temporal areas, cingulum; *D*, between frontal and temporal areas, fasciculus uncinatus; *E*, between occipital and temporal areas, fasciculus longitudinalis inferior; *CN*, caudate nucleus; *OT*, optic thalamus.

FIG. 18.—The Projection Tracts joining the Cortex with Lower Nerve Centres. Sagittal section showing the arrangement of tracts in the internal capsule. *A*, Tract from the frontal lobe to the pons, thence to the cerebellar hemisphere of the opposite side; *B*, motor tract from the central convolutions to the facial nucleus in the pons and to the spinal cord; its decussation is indicated at *K*; *C*, sensory tract from posterior columns of the cord, through the posterior part of the medulla, pons, crus, and capsule to the parietal lobe; *D*, visual tract from the optic thalamus (*OT*) to the occipital lobe; *E*, auditory tract from the intergeniculate body, to which a tract passes from the VIII. N. nucleus (*J*) to the temporal lobe; *F*, superior cerebellar peduncle; *G*, middle cerebellar peduncle; *H*, inferior cerebellar peduncle; *CN*, caudate nucleus; *CQ*, corpora quadrigemina; *Vt*, fourth ventricle. The numerals refer to the cranial nerves.

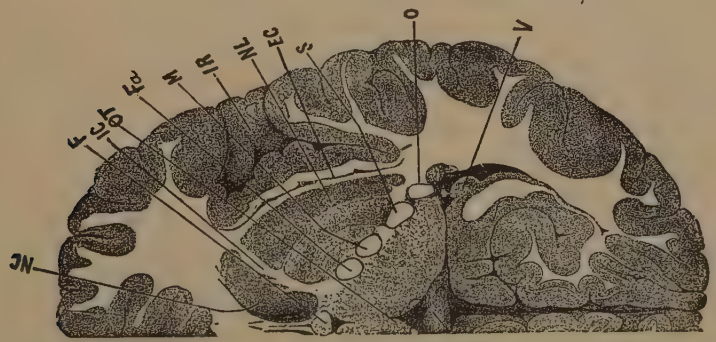


FIG. 19.—Horizontal Section through Right Hemisphere showing the Internal Capsule and Basal Ganglia. *F*, Fornix; *NC*, caudate nucleus; *OT*, optic thalamus; *NL*, lenticular nucleus; *IR*, island of Reil; *EC*, external capsule; *V*, lateral ventricle; *IC*, int. capsule, ant. part; *Fα*, facial tract; *M*, motor tract to limbs; *S*, sensory tract; *O*, visual tract, all in the posterior part of the capsule.

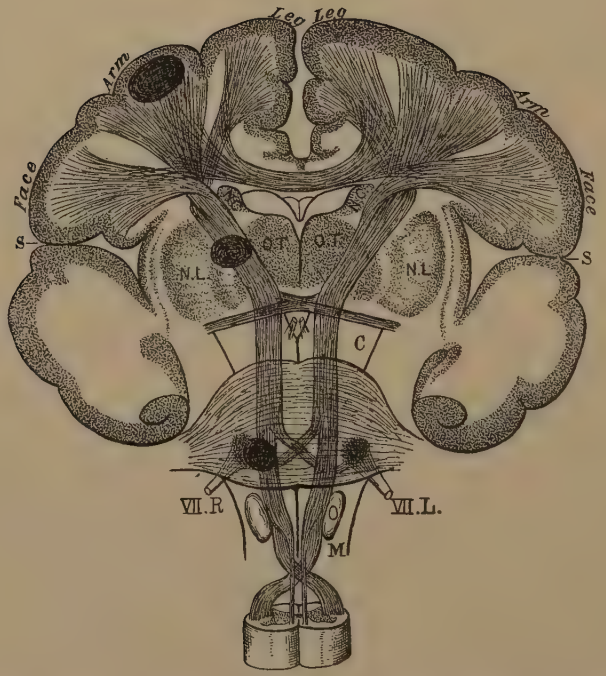


Fig. 20.—The Motor Tract. *S*, Fissure of Sylvius; *NL*, lenticular nucleus; *OT*, optic thalamus; *NC*, caudate nucleus; *C*, crus; *P*, pons; *M*, medulla; *O*, olivary body. The tracts for face, arm, and leg gather from the lower, middle, and upper thirds of the motor area, pass into the capsule and through the crus and pons, where the face fibres cross to the opposite VII. N. nucleus, while the others pass on to the lower medulla, where they partially decussate to enter the lateral column of the cord, the non-decussating fibres passing into the antero-median columns. Lesion in cortex causes monoplegia; in capsule, hemiplegia; in pons, alternating paralysis.

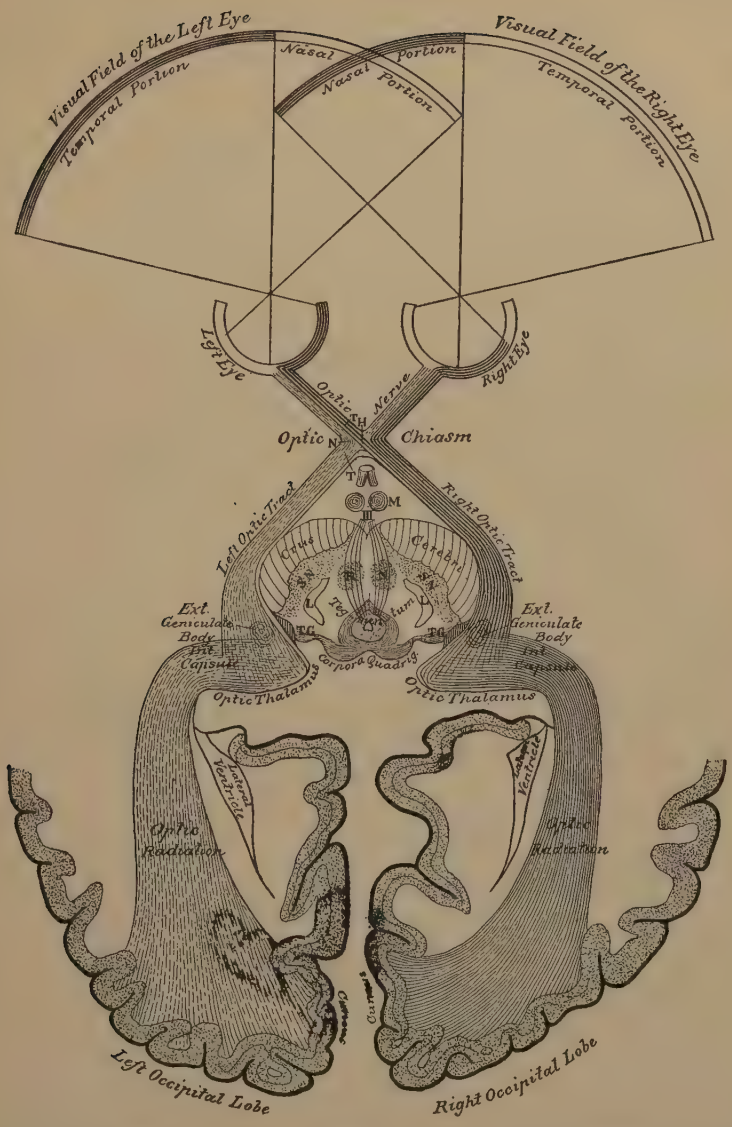


FIG. 21.—The Visual Tract. The result of a lesion anywhere between the chiasm and the cuneus is to produce homonymous hemianopsia. *H*, Lesion at chiasm causing bilateral temporal hemianopsia; *N*, lesion at chiasm causing unilateral nasal hemianopsia; *T*, lesion at chiasm causing unilateral temporal hemianopsia; *SN*, substantia nigra of crus; *L*, lemniscus in crus; *RN*, red nucleus; *III*, third nerves.

FIG. 22.—Diagram of the corpora quadrigemina anterior, *CQA*, showing their connections. On the right c the figure the superficial and deep masses of gray matter are shown. *Pulv*, Pulvinar of the optic thalamus; *Pr* posterior nucleus of the optic thalamus lying between the corpus geniculatum externum, *cge*, and the corpu geniculatum internum, *cgi*; *IC*, internal capsule; *F*, fillet; *RN*, red nucleus of the tegmentum; *PED*, peduncle c the cerebrum; *Sn*, substantia nigra; *OT*, optic tract; *X*, optic chiasm; *II*, optic nerve; 1, 2, fibres from th retina to the pulvina of the optic thalamus; 1, centripetal; 2, centrifugal; 7 and 8, fibres between the opti thalamus and the occipital cortex; 3 and 4, fibres between the retina and the corpus geniculatum externum; and 10, corresponding fibres to the occipital cortex; 5 and 6, fibres between the retina and the corpus quadrigem num anterior; 11 and 12, corresponding fibres to the occipital cortex—for the sake of distinctness the decussatin optic fibres only are traced; 13, cell of the superficial gray matter of the *CQA* passing to the nucleus of the thir nerve (16); 14, cell of the deep gray of *CQA* passing to third nerve nucleus; 15, cell of the deep gray of *CQ* sending fibre into the fillet; 17, fibre from the red nucleus terminating about 14; 18, fibre from the fillet termina ting about 13; *OL*, occipital lobe of the brain, with its cortex, containing both cells and terminal brushes of th visual tract.

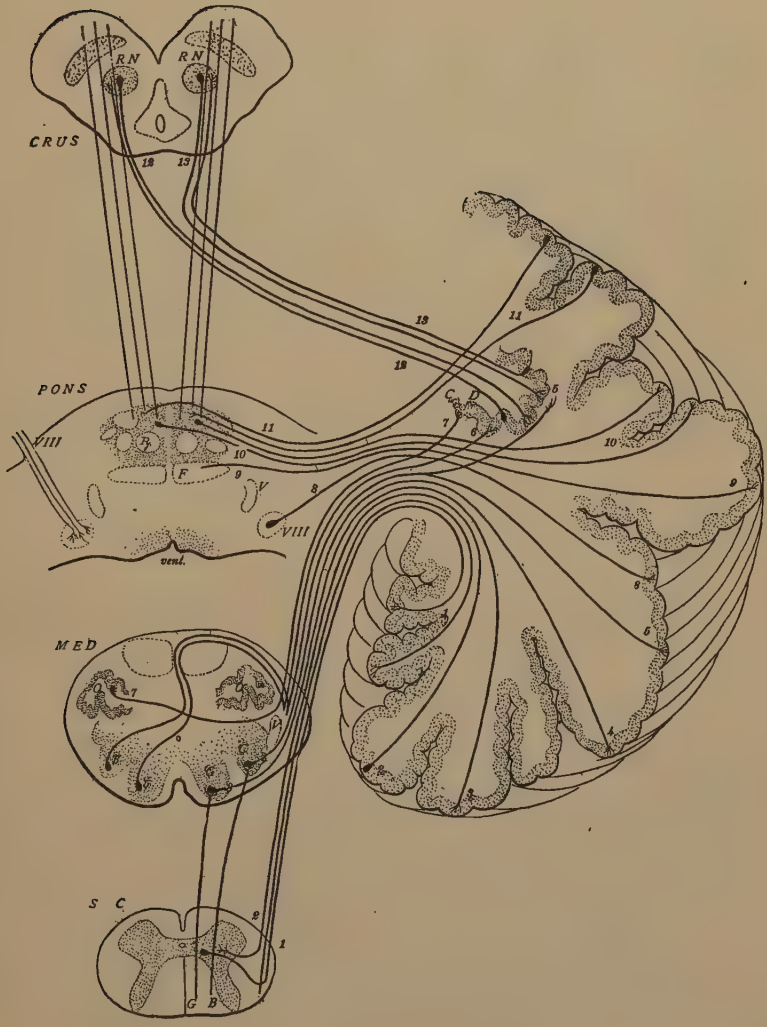


FIG 23.—Diagram to show the connections of the cerebellum. *SC*, Spinal cord; *G*, column of Goll; *B*, colun of Burdach; *MED*, medulla oblongata at sensory decussation; *O*, olive; *G*, nucleus gracilis; *C*, nucleus cuneat *V*, fifth nerve; *Pons*, pons Varolii; *VIII*, eighth nerve and its nucleus; *F*, fillet; *Py*, pyramids; *CRUS*, cr cerebri; *RN*, red nucleus of tegmentum; *CD*, corpus dentatum of cerebellum; 1 to 13, various tracts enteri cerebellum from spinal cord, medulla, pons, and crus. For description see text.

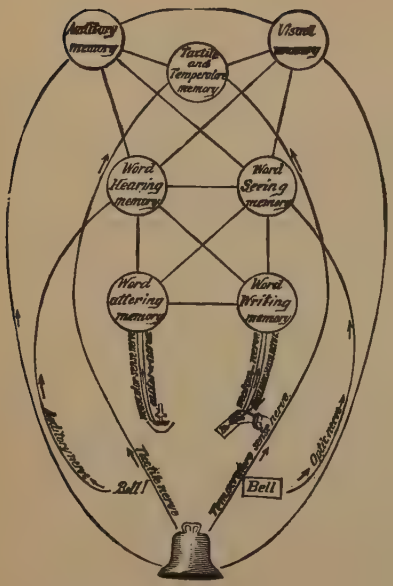


Fig. 24.—Diagram to illustrate the Concept "Bell" and to show the Varieties of Apraxia and Aphasia. The memory pictures are relics of past perceptions received through different senses. The association makes up the mental image bell. The word image is made up of the memories of the sound and appearance of the word, and of the uttering and writing effort memories; these are joined together. The mental image and the word image are also joined with one another, making up the concept "bell."

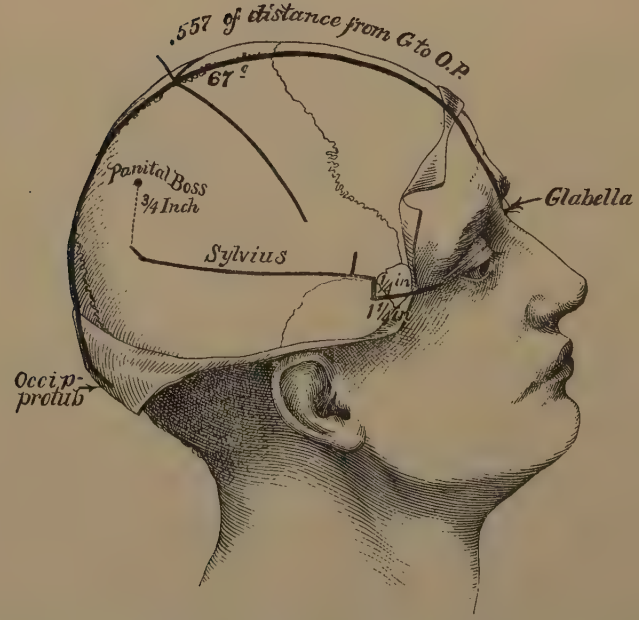


Fig. 25. Diagram to show the Relation of the Skull and its Sutures to the Fissures of Rolando and Sylvius.

FIG. 25.—Diagrammatic Representation of a Section through the Cerebellar Cortex. *I*, Molecular layer; *II*, granular layer; *III*, white matter; *P*, Purkinje cell with its neuraxon, *p*, entering the white matter; *S*, small stellate cells of molecular layer; *B*, large stellate cells with basket fibres, *b*—these basket fibres surround the body of the Purkinje cell shown in dotted outline; *G*, cells of the granular layer with long, straight neuraxon, *g*, ascending to molecular layer, and there bifurcating to become tangential fibres—these fibres run at right angles to the plane of section of the plate; *M*, moss-like termination of white fibres, *m*, entering the cerebellum from without; *H*, large Golgi cell of the second type with dendrites in both granular and molecular layers and neuraxon dividing and subdividing within granular layer; *I*, terminal filaments and fibres, *i*, entering the cerebellum from without and ending around the branches of the Purkinje cells.

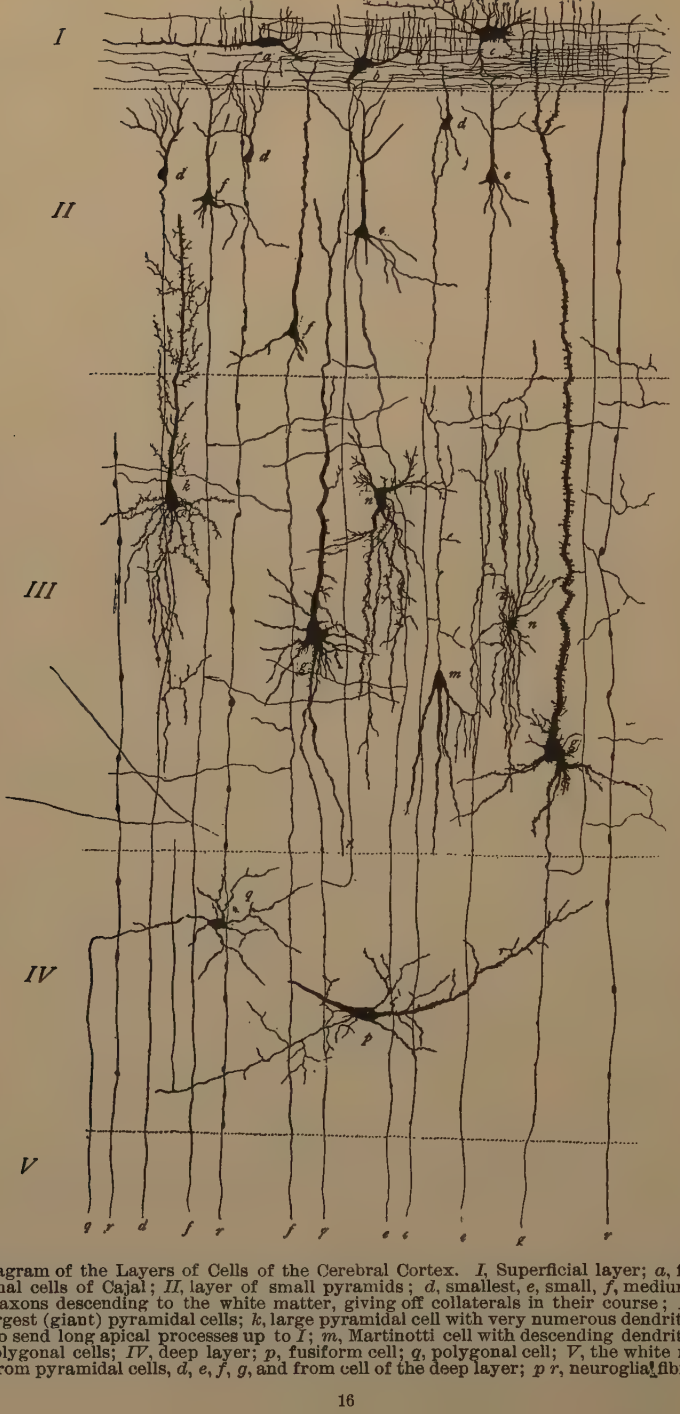


FIG. 26.—Diagram of the Layers of Cells of the Cerebral Cortex. *I*, Superficial layer; *a*, fusiform, *b*, tri gular, *c*, polygonal cells of Cajal; *II*, layer of small pyramids; *d*, smallest, *e*, small, *f*, medium-sized pyram with their neuraxons descending to the white matter, giving off collaterals in their course; *III*, layer of la pyramids; *g*, largest (giant) pyramidal cells; *k*, large pyramidal cell with very numerous dendrites; all pyram cells are seen to send long apical processes up to *I*; *m*, Martinotti cell with descending dendrites and ascend neuraxon; *n*, polygonal cells; *IV*, deep layer; *p*, fusiform cell; *q*, polygonal cell; *V*, the white matter contain the neuraxons from pyramidal cells, *d*, *e*, *f*, *g*, and from cell of the deep layer; *p r*, neuroglia fibre.

LECTURES

UPON

DISEASES OF THE MIND

DELIVERED AT THE

COLLEGE OF PHYSICIANS AND SURGEONS

NEW YORK

BY

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PREFACE.

THIS synopsis of lectures upon diseases of the mind has been prepared for the use of the students of the College of Physicians and Surgeons.

In the presentation of the subject the method of Krafft-Ebing in his "Lehrbuch der Psychiatrie" has been followed.

The attempt is made to study mental processes in connection with the symptoms of insanity, and to show that the action of a deranged mind differs not so much in kind as in origin and degree from that of the sane mind.

The forms of insanity are then described, Meynert's classification being adopted. The causes which lead to mental disease, the data which bear upon its prognosis, and the proper means for the care of the patient, are considered. And, finally, some account is given of the proper method of examining a person suspected of lunacy, and of the legal measures necessary in New York to his commitment to an asylum if insane.

To those to whom Krafft-Ebing's work and Meynert's lectures (which have unfortunately not yet been translated) may be inaccessible, the works of Bevan Lewis, "A Text-book of Mental Diseases," of Mercier, "Sanity and Insanity," of Kirchhoff, "Handbook of Insanity," of Spitzka, "Insanity," and of William James, "Psychology," are recommended.

OCTOBER, 1897. M. ALLEN STARR.

INSANITY.

THE study of insanity implies a knowledge of the normal action of the mind, and includes a consideration of abnormal mental action, of the symptoms which indicate mental derangement, of the forms which that derangement may take, of the causes which produce it, of the prospect of recovery, and of the appropriate care of the sufferer.

The physical basis of all mental action is the cerebral cortex with its association fibres; for its extent, thickness, and blood supply determine the mental capacity of the species, of various races, and of single individuals; and its disease or defect causes insanity or idiocy.

The functions of the cortex are: (a) To receive impressions from the sensory organs, which the mind knows as conscious perceptions. (b) To group these impressions into a unit, which the mind knows as a concept. (c) To retain the concepts, so that the mind can recognize and recall them in memory, or form new combinations between them in imagination, or group them systematically in logical thought. (d) To give expression to thought in speech and in action. (e) To feel the emotional excitement attendant upon these mental acts. (f) To exercise self-control over all mental action.

When small areas of the cortex are diseased, one or more of these functions may be suspended; this alone does not imply insanity. But when the entire cortex is affected by temporary changes of nutrition, or of circulation (as in starvation or intoxication), or by processes of disease, the harmony of interaction of its various parts is disturbed, and delirium and insanity result. Mental symptoms are then produced.

It is a relic of the old superstition that the insane are possessed by the devil to consider mental symptoms as mysterious. The processes of mental action in a deranged mind do not differ in kind from those in a sane mind. The depression of grief and of melancholia, the excitement of great success and of mania, the resentment at injustice, real or imaginary, are identical in kind. The differ-

ence between sane and insane mental action lies in the *origin* of the mental process, and in the *degree of its intensity*. In health, thought, emotion, and action are determined by external circumstance or by logical mental processes starting from true premises, and the resulting feeling or action is proportionate to the thought or motive. In mental disease thought, emotion, and action are determined by internal causes, unexplained by external circumstance, often without actual premises, and out of proportion to the supposed cause. It is therefore the origin and degree rather than a new kind of mental action which are to be investigated in the study of insanity.

Thus two men may show signs of distress or of despair: in one an adequate cause is found in family affliction or financial ruin; in the other no reason can be ascertained excepting the hypothesis of disease. Two men may perform a theft: in one, poverty combined with opportunity and a low moral standard will explain the act; in the other no motive can be found, and the only excuse given may be that of a sudden overwhelming desire to steal only assignable to disease. The manifest emotion or action in both is the same. The difference which makes the latter insane in each case is the origin of the feeling or act.

So long as our ideas, feelings, and actions are in harmony with our education and surroundings, and the reaction between external agents or influences and the state of mind is in natural proportion, so long are we sane. For all ideas must have a logical basis, all feeling must have an adequate cause, and all action must have a sufficient motive, in a sound mind. And hence it is in the origin of mental action rather than in its process that insanity usually begins. Thus, an idea without reason, an emotion without cause, an act without motive, are manifestly insane.

THE SYMPTOMS OF INSANITY.

These may be conveniently studied according as they indicate disturbance in

(*a*) Conscious perception.
(*b*) Logical thought.
(*c*) Emotional feeling.
(*d*) Voluntary action.
(*e*) Self-consciousness.

(*a*) DISTURBANCE OF CONSCIOUS PERCEPTION.—Conscious per-

ceptions are usually the result of cortical action set up by impressions received from the sensory organs through the intermediate subcortical centres.

Thus the impact of light upon the eye sends an impression through the optic nerve to the optic thalamus and corpora quadrigemina, where reflex results, such as convergence of the eyes toward the light and action of the pupils, are produced; and thence through the visual tract to the cortex, where the light, as well as the incidental ocular movements, come before consciousness, which then by an act of comparison between the sensation received and other prior sensations remembered, perceives the light as light of some particular kind in a particular place. These perceptions may be rendered more acute or may be blunted by disease. Thus in meningitis light may be painful at first, but later the patient may actually become blind. It is a mental power to increase the acuteness of perception by attention. Thus sounds which otherwise would pass unnoticed may be heard if we listen.

Conscious perceptions leave behind them a trace which we can revive in memory. And by imagination it is even possible to call to mind so vividly the mental pictures which remain as traces of former perceptions that we almost reach the point of reviving the image so clearly as to suppose that we see it. Galton calls this the power of mental imagery. It is to the visual power what the power to silently hum a melody is to the auditory power.

When attention is strained and expecting a perception a slight sensation may give rise to a false perception; or a misinterpretation of the sensation perceived. Thus the face of a stranger may be mistaken for that of a friend; the ringing in the ear produced by a bit of wax may be interpreted as a chime of bells; a white gravestone on a dark night may be thought to be a ghost. *An illusion is a real perception produced by a sensation falsely interpreted by the mind.* Such false interpretation is a common symptom of insanity. The tick of a clock is thought to be a voice addressing the sufferer. The reflection of the light from a shining object in the room is thought to be a face of angel or devil appearing to him. A stranger is supposed to be some friend risen from the dead.

In a sane man the misinterpretation is easily corrected by other considerations. He picks the wax from his ear; he satisfies himself that the object is not a ghost. The insane man accepts the misinterpretation without investigation and believes in its truth. He may make it the basis of a train of thought and action without seek-

ing to establish its validity. Thus the illusion that a voice commands him not to eat may lead to an attempt at suicide by starvation. The act is then insane because based on an illusion which is the product of a diseased mind. The indisposition to correct illusions is in itself a grave symptom of insanity.

But conscious perceptions are capable of being produced without actual sensations from without. Any process which can excite the cortex to activity can cause a conscious perception, probably by exciting the physical basis of memory pictures to such a degree as to equal in intensity the stimulus of the original sensation and thus produce a mental perception. A blow on the head gives a sensation of light, opium or cannabis indica causes false perceptions of faces and figures. In delirium false perceptions of wholly internal origin are common. And in insanity such *false perceptions without any ascertainable external sensational origin occur. These are called hallucinations.* Such perceptions are mental revivals of past perceptions, for blind men never have hallucinations of sight and deaf-mutes never have hallucinations of hearing. Their occurrence implies intense activity of cortical function—possibly under great excitement (*e.g.*, Luther seeing the devil), usually in disease (*e.g.*, Joan of Arc seeing the Virgin). In a sane man hallucinations are at once corrected. Thus the zigzag lines of light seen in an attack of migraine are never thought to be external to the eye. The insane man accepts the perception as real and believes in its truth. Consciousness has no means of determining whether a perception is false or real excepting by a process of reason. An hallucination cannot be denied but must be explained. The insane man does not explain it, and because he does not he is insane. When an insane person begins to explain and correct his illusions and hallucinations his sanity is returning.

Illusions and hallucinations in any individual may be stable, that is, always of one kind; or changeable, of many kinds; they are usually intermittent, rarely constant. They may be an early symptom of insanity; thus a patient with periodical mania saw a gray bird flying around his head for some days prior to each attack. They may continue through the disease. They are not always present. They may occur in any variety of sensory perception, as visions, as voices, as smells or tastes (especially when the tongue is coated), as sensations of touch (hyperæsthesia of the arm leads to the delusion that the arm is glass), and as sensations of muscular sense or movement (patients think they can fly). They may sur-

prise the patient and lead to delusions or to insane acts, or to great emotional excitement. Insults heard may cause rage and lead to violence, or cause grief, despair, and suicide. A woman seeing several strange men about her constantly is greatly distressed at the insult of their presence.

Hallucinations and illusions, if not confessed by the patient, may often be detected by noticing sudden changes of facial expression, anxious listening or looking about, or uncalled-for remarks as if in answer to questions heard.

Conscious perception is necessary to recognition of objects and persons and to the proper reaction of the mind to its surroundings. A loss of the power of conscious perception may occur in disease and is a symptom of insanity. The patient is then like a baby who has never learned the use of things; he loses all interest in things; he mistakes one for another—stones for diamonds, tinsel for gold; he loses his way, forgets his relatives, becomes helpless and *demented.* The loss of the power of conscious perception occurs in the terminal stage of insanity, rarely early in its course. A defect of conscious perception due to inattention, the mind being so absorbed with its own train of thought or state of feeling as to neglect all surroundings, as in melancholia or stuporous insanity, occasionally occurs, but does not imply loss of actual power. Hence in such states sudden recovery is possible when the attention returns.

(*b*) Disturbance in Logical Thought.—The mind is constantly combining the conscious perceptions or their memories into ideas, which are either concrete or abstract, the latter being handled by the aid of language. This play of consciousness is not at random, but always normally along the lines of association which have originally been established by simultaneous perception of associated objects and subsequently strengthened by repeated use.

Thus the idea "bell" is made up of the memories of the shape, sound, and appearance of numerous special bells seen and heard, and of the word "bell" spoken, heard, written, or read, each of these memories having a separate location in the cortex, all being joined together by association fibres and any one being recalled to mind when any other is brought into consciousness by a perception of the object or its name. The idea "charity" is made up of numerous memory pictures of acts of benevolence or of descriptions of such acts, the multitude of thoughts connected with it being handled easily by the aid of the word; language being the necessary basis for all abstract thought.

The action of the mind in passing from one subject to another along definite lines of association is logical thought.

The process of association of ideas may be affected by disease. Thought may be delayed, so that mental action is manifestly sluggish and painful. Thus the replies to questions are slow in a melancholic, and he cannot be hurried. Such slow thought makes time seem endless and in itself gives great distress. The process of association may be arrested so that a question no longer leads the mind to its answer, a sensation no longer calls up associated ideas; the condition is then one of stupor. On the other hand, thought may be hastened by disease which excites the brain, and thus associations may become abnormally rapid, so that a single perception may start many unfamiliar and unexpected ideas, which occur too rapidly to be expressed. Thus the rapid thought of a maniac outruns his language and leads to incoherent talk, speech being too slow for his ideas. This rapid thought makes the experience of an hour seem that of a year. Time is measured by the rapidity of mental processes, each association normally taking one-eighth of a second. Hence in disease the time sense is impaired.

In insanity ideas may be suddenly brought before the mind from internal causes without logical associational process. Such *compulsory ideas* resemble in origin hallucinations. They arise spontaneously and cannot be referred to any process of thinking. They surprise and alarm the patient. Examples: sudden fear of friends, idea that clothes are contaminated, impulse to swear, to throw one's self off a height, desire to touch certain objects, to steal. The persistence of such ideas may lead to insane acts or to the formation of delusions, if they are not recognized as of internal origin. *Delusions are mistaken ideas without sufficient logical foundation, accepted as true.*

A sane man may have a mistaken idea, *e.g.*, seated in a stationary railway train the movement of a passing train combined with the expectation of starting leads to the delusion that we are moving. This is corrected by glancing at the other side. A man unfortunate in business usually explains it rationally by his circumstances, or his incapacity, never, unless insane, by the hypothesis of a widespread conspiracy among strangers to secure his ruin. The lack of logical foundation for such an idea and the acceptance of the idea without question make it a delusion.

Delusions may arise from reflection upon mistaken premises and then be logical. Thus Joan of Arc, having seen the Virgin and re-

ceived commands from her (hallucinations), considered herself inspired for a mission. A man considers that he has committed the unpardonable sin because his thoughts are all depressing, being governed by his emotional state, which is thus the primary insane origin of the delusion. Delusions may arise from reflection on a valid premise but be illogi al. Thus pain in the stomach gives rise to the delusion that an animal is living in the abdomen. Many delusions are theories which patients invent to account for bodily sensations. Lastly, delusions may arise spontaneously, like compulsory ideas and hallucinations, being entirely of internal origin—examples, the delusion of perscution, the delusion of grandeur. They are often thought over, elaborated, brought into relation with every event of life, and so finally become the dominant thought of the person, controlling his entire mental action without necessarily impairing any of its processes (so-called monomania). Many delusions are illogical and absurd and transient. Others are well worked out, are systematized, and the acts and feelings are the natural result of the delusion.

The character of the delusion is influenced by the surroundings, life, education, and habits of the person. Brokers get delusions of wealth or poverty; clergymen get delusions of damnation for infidelity; mechanics discover perpetual motion. In Europe insane think they are noble; in America they think they should be appointed to office (Guiteau). Formerly witchcraft was thought to influence patients, now electricity is the agent. The existence of bodily disease may influence the delusion. Thus pleurisy gives rise to the idea that enemies are sticking knives into the patient's side. Persistent delusions referred to any organ should lead to suspicion of actual disease there. Lastly, the form of insanity often determines the delusion. Thus in religious melancholia the patient believes that he has committed the unpardonable sin; in chronic alcoholic insanity the delusion of conjugal infidelity is usual; in paresis the patient believes himself well, strong, and wealthy; in paranoia the delusion of persecution by a conspiracy is constant. There are very few insane persons who are free from delusions, and hence this is the most important symptom of insanity.

(*c*) Disturbance of Emotional Feeling.—All mental action is attended by pleasure or pain, either distinct or indistinct, which varies in degree and complexity in accordance with cerebral nutrition. There are times when we are easily amused or excited, there are times when we are easily depressed; temperament determines

the prevailing emotional state or the tendency to emotional reaction to surroundings. In a normal man spontaneous variations of emotion occur within wide limits, so that the same subject may at one time give pleasure, at another pain, *e.g.*, interest in a lecture or a play. In insanity a greater emotional variability may be present, so that emotions are more intensely felt and more readily produced, or they may be unusually dulled. There may be an undue feeling about trivial matters, *e.g.*, fear of dirt leads the patient to wash his hands constantly; he shows great excitement over unimportant events. There may be lack of any feeling, because the mind is constantly absorbed with a delusion, *e.g.*, the patient becomes indifferent to family and business affairs, so that serious financial trouble does not seem to affect him; or he may become dirty. Patients often complain that they can take no interest in anything. There may also be a spontaneous emotional condition of very intense kind, dominating all mental action, such as great depression or great exaltation, due entirely to disease.

Depression is characteristic of melancholia. Nothing interests or pleases, and even thought is painful and action disagreeable. Such patients have intense anxiety without being able to assign a cause. The depression may lead to the formation of a delusion to explain the depression, *e.g.*, idea of a fatal disease.

Exaltation is found in mania and in paresis—a sense of great power and ability leading to foolish boasting or to reckless acts. This may give rise to delusion of inspiration or grandeur.

Primary emotional states must be distinguished from secondary states due to logical thought, on the basis of delusions or hallucinations. The latter are usually less prominent and less intense. The existence and character of emotional states are shown by facial expression and action.

An actual perversion of emotion may occur in severe cases, where things which should disgust, please, *e.g.*, patient drinks urine; sexual perversion.

(*d*) Disturbance in Voluntary Action and Conduct.—Voluntary action is the outcome of sensation, thought, and emotion, controlled by conscious motives. There is an unconscious, unwilled activity accompanying all mental action, known as muscular tone, and shown in facial expression, attitude, and automatic movement. In disease the activity may be weakened from perverted sensations, slow and difficult thought, or deficient emotion, in which case action seems useless. Thus a melancholic is often inactive and looks sad.

Or the activity may be increased, an unwonted sense of freedom may be felt from excitement in all mental functions. Thus a maniac is never quiet, is in motion and noisy and ambitious and looks eager. If this unconscious activity is repressed by disease, a nervous stress results which often causes pain and leads to final relief in maniacal outbursts. Hence the occasional agitation in melancholia. Spontaneous or impulsive acts occur in insanity, analogous to hallucinations, delusions, and primary emotional states, not traceable to conscious motives and certainly without choice—example: constant activity of a paretic, sudden act in response to sensation, or hallucination, or emotion, *e.g.*, rape, murder, suicide, aimless movements of body of anxious melancholic, repetition of same phrase in katatonia.

Voluntary action may also be affected in insanity. The deranged mind may not weigh motives properly, or may be deceived by delusions, or may yield to sudden impulses, and hence the patient's conduct is insane, *e.g.*, extravagant expenditures of paretic; homicide by paranoiac under delusion of persecution; kleptomania or pyromania. Or the weakened mind may be unable to choose between conflicting motives, and be constantly questioning, and hence fails to decide, *e.g.*, insanity of doubt.

Responsibility is impaired when it can be proven—

(1) That an act was the result of hallucination, delusion, uncontrollable emotion, or morbid impulse due to disease.

(2) That the motives for and against the act could not be properly weighed on account of weakened intellectual power.

(3) That the import of the act could not be realized from impaired memory or consciousness.

As a plea in defence of criminal acts other insane acts and other symptoms of insanity besides the one in question must be proven.

(*e*) Disturbance of Consciousness.—Mental action is partly conscious and partly unconscious—that is, there are degrees of distinctness in the various subjects occupying consciousness, *e.g.*, while interested in a play one is only partly conscious of the seat being uncomfortable, the theatre hot, and the head aching from the bad air. An act is only distinctly conscious when attention is directed to it and a memory of it remains. Many of our transient moods, many automatic acts, and our sensation of well or ill-being are largely sub-conscious, *i.e.*, go on without attention.

The range of consciousness includes consciousness of external

things and of internal thoughts, and there is normally a balance between these. If the attention is concentrated upon internal thought the external field of consciousness is correspondingly limited as the internal field widens. Introspection may exclude external impressions. Hence, a hypochondriac has few interests outside himself and becomes conscious of sensations usually unnoticed, *e.g.*, the heart-beat. A melancholic patient has his entire attention devoted to his own state of feeling, and is not affected by surroundings. It does not help him to give him diversion.

Consciousness may be weakened in insanity; thus, the duration of impressions may be too short for the slow attention and memory, and the continuity of thought may thus be lost—*e.g.*, in dementia a patient may get lost in his own house, or if away from home be unable to tell who he is or where he lives; may lose his personality. A sort of somnambulistic state may be present, or a state of double consciousness.

The personality is the sum of continuous acts of self-consciousness. A change of personality may be a symptom of insanity, the insane ideas and emotions being so foreign to the normal man that his insane character and disposition cannot be brought into harmony with his former personality in consciousness, and so he declares that he is another person, *e.g.*, a king, Christ.

Finally, consciousness may be almost suspended for a time, *e.g.*, in stuporous insanity, in which a patient has, after recovery, no memory of weeks of existence.

. . . . . .

The symptoms of insanity are thus seen to be variations in degree of normal mental processes, often of internal origin. They are not mysterious, nor do they differ in kind from ordinary states of mind.

THE PHYSICAL SYMPTOMS OF INSANITY.

1. Sensory disturbances, such as anæsthesia, hyperæsthesia, or pain, may occur, and are often the foundation for illusions and delusions.

2. Reflex action may be affected in various ways—lessened because of increased inhibitory attention, increased because of weakened inhibition or because of direct irritation from disease. Thus

Christian science does no goods in any mental disease.

the pupils may vary, being often dilated in melancholia and contracted in mania or paresis.

3. Motor disturbances are noticed both in the state of muscular tone and in the power of voluntary action. Facial expression and attitude, character of movements, and of speech, and of writing, and peculiarities of conduct, are often valuable symptoms in differential diagnosis.

4. Vaso-motor, trophic, and nutritive changes are commonly found in insanity. In states of depression the circulation is impaired, the pulse is small, the skin pale. In states of excitement the face is flushed, the surface hot. In both weight is lost and digestion is much impaired. All emotion reacts upon the bodily functions.

5. Insomnia is the most prominent physical symptom of insanity. Headache and vertigo are rare. Convulsions occur in paresis sometimes, and paralysis in the last stage.

THE FORMS OF INSANITY.

Melancholia.

Melancholia is a spontaneous, painful, emotional state with depression of all the mental and nervous functions.

It begins gradually. The patient feels despondent; loses interest in things about him—in his family, business, religion, and amusements; prefers seclusion, as all impressions become painful; becomes conscious of inability to think clearly or quickly, to read or to study, to enjoy life, or to do his work; and feels disinclined to exert himself. From time to time a sudden, unexplained anxiety, a fear of impending evil with palpitation of the heart comes over him, and he dreads insanity and thinks of suicide.

This condition may remain for some time as a *simple melancholia*, or be the prodromal stage of mania.

If the depression becomes painfully intense and continuous it leads to delusions. In health the prevailing emotion colors our interpretation of events. So in disease. In melancholia delusions are always of a self-condemnatory character. The patient thinks that he has offended the Deity, has committed the unpardonable sin, and blames himself for the most trivial acts and thoughts as well as for ordinary transgressions, and will not admit that he can repent or

be pardoned. Or he thinks that he is an outcast from society, that he has no friends and no home, is too vile to associate with other men, is the greatest of criminals, and that nothing remains except suicide; that he is not worthy of care or shelter, and that it is useless to attempt any treatment, for it is folly to consider his state one of disease. He believes it a just punishment for his sins, but so painful that death seems desirable.

This condition may remain for some time as *melancholia with delusions.*

In severer cases hallucinations and illusions, especially of hearing, begin, and are of a character to increase the delusion and alarm the patient. The voice of God is heard condemning him. The devil tempts him by every form of evil suggestion and accusation, even taunts him with fear of suicide. Voices call him the chief of sinners, the worst of mortals, and command him to kill himself, or tell him he is to live forever in misery. Hallucinations of other senses are added, imps are seen about him and fearful shapes threaten. His bowels are being eaten by an animal within, his organs are being destroyed by electricity; he can taste nothing because of foul odors. The hallucinations and delusions are usually of a religious or hypochondriacal character.

In this condition the distress may at times become too great to be borne in silence or in quiet; the patient becomes restless, excited, violent, noisy, crying out in despair and making many attempts at self-mutilation or suicide. (*Melancholia with agitation.*)

Finally, if suicide is prevented, he may sink into a state of stolid stupor, be wholly absorbed in his mental agony, lose his personal identity, be devoid of energy, and neglect to eat, and even disregard ordinary cleanliness. He sits or stands wherever placed, silent, motionless, stupid. (*Melancholia with stupor.*) Even in this state sudden wild attempts at suicide may be made.

Whatever the degree of melancholia recovery is probable and occurs slowly, the symptoms gradually subsiding; the remissions in the state of depression, which indicate beginning recovery, occur toward evening.

In melancholia the sensory perception is slow and imperfect, partly because attention is not paid to the surroundings, but is concentrated upon the emotional state. The processes of thought are delayed, association time being perceptibly increased, and all thought is attended by a sense of weariness and effort. The play of emotion is hampered by the depression present, nothing seems to

please or to divert, and the intensity of depression is out of all proportion to any possible cause. Voluntary action becomes disagreeable and even impossible, the necessary stimulus in motive being wanting, choice being difficult and effort seeming impossible. The instinctive acts are slowly performed, there is a loss of muscular tone, and feeling of impaired power; the automatic expressions of distress, such as crying, are at first suppressed but later may become violent—such as constant movement, tearing the hair, beating the body, groaning, pacing the floor for hours. Consciousness is clear throughout and memory of the disease remains, but consciousness is concentrated upon internal mental action and disregards the external world.

The physical symptoms are well marked. The facial expression is that of distress or despair and lacks all sign of interest. Nutrition is rapidly impaired, as indigestion and constipation are present, and weight is lost. The patient is sleepless, and suffers from precordial anxiety, and palpitation. The pulse is small and tense. The pupils are dilated. The reflexes are diminished.

The course of the disease is as follows: a gradual development of the mental and physical symptoms to a greater or less degree of intensity in different cases, a stationary period of some weeks during which there is constant danger of suicide, and then a termination in (1) recovery in sixty per cent., which is more or less rapid according to the severity of the disease; or (2) the development of mania, which runs its course; or (3) the development of chronic insanity with fixed depressed delusions, and finally dementia after a period of years. Relapses are not uncommon and a return of the disease occurs in ten per cent. of the cases. Some patients commit suicide, others die of exhaustion, others of complicating pulmonary or intestinal diseases.

The prognosis is favorable, as sixty per cent. recover. The duration in the cases which do recover is six months or less in forty-five per cent., under a year in thirty-seven per cent., over a year in eighteen per cent. The first sign of recovery is a gain in weight.

The treatment of light cases without delusions consists in increasing the nutrition by frequent feeding, with the aid of tonics, stimulants, laxatives, and aids to digestion, and the use of an excess of fluids, especially milk and beef extracts (forcible feeding may be necessary); the use of long warm baths followed by short cool douches to head and back, massage, and out-of-door exercise with-

out fatigue; the use of vaso-dilators and cardiac stimulants, viz., nitroglycerin or chloral or opium with strophanthus, sparteine, or alcohol; and the use of hypnotics, viz., chloral, amylen hydrate, chloralamide, or sulphonal.

The treatment of severe cases with exhaustion demands absolute quiet and rest in bed. In every case precautions against sudden impulses to suicide must be taken from the first.

It is better to keep a melancholic asleep than to attempt to relieve his distress by diversion or anything which demands a mental effort which he cannot make.

Mania.

Mania is a spontaneous exalted emotional state with increased activity of all the mental and nervous functions.

It begins gradually, as a rule, with a prodromic period of depression. It occasionally begins suddenly, *e.g.*, after childbirth. After the period of depression, in which the symptoms are those of simple melancholia, the patient passes into a state of exhilaration and eager excited mental activity, in which perception is very keen, the association of ideas unusually rapid and numerous, memory vivid, thought quick and easy, the emotional state one of pleasure easily aroused, activity great, movement and speech free and spontaneously agreeable, a sense of power and force is felt, consciousness being clear and life happy. He goes about among his friends showing unusual wit and social instincts, takes long journeys, joins in new enterprises, spends money beyond his means. The physical functions are also increased, the patient looks well; all the appetites, especially the sexual, are heightened, but the state of mind does not permit of sleep, and the constant activity leads to loss of weight.

As the condition goes on the torrent of ideas and impulses becomes greater, and all checks upon action—intellectual, social, or moral—seem removed. The patient yields to all impulses without restraint; shouts and sings and dances; any opposition causes irritability of temper and combativeness; and acts of violence, profane extravagant language and maniacal action are indulged in at slight provocation. The patient affirms that he never has felt so well, so happy, so confident of his powers, and so brilliant as he feels in this state of *simple mania*. Such a state may be a stage in the course of "circular insanity" (alternation of melancholia and mania), or of

Wet packs temp. 85° - for 30 min to 1/2 h
Follow up - rub up H_2O - at 85 - & then br
brush. High enema at 80 to 90° up -
Borax ʒi to Water Oii. Retain enema as
long as possible, at least 1 hr.
Starr thinks it due to autoinfection - he
keep intestines clean - salol & cas
tor oil.

hysterical insanity, or it may occur in attacks which subside (periodical mania), or it may go on to *mania with delusions and frenzy.*

For the rapidity of thought may lead to a confusion of ideas in which reasoning is abandoned and actions are done in response to perceptions without consideration of motive or responsibility. The sight of a knife leads to an attempt at murder, the sexual impulses lead to rape. Soon hallucinations and illusions appear (occasionally they occur at the outset), increasing the mental excitement, for the mind does not stop to correct mistaken perceptions. They are of all kinds in all senses, especially of sight, and usually are agreeable, or, if not, are interpreted favorably, being colored by the emotional state. Even unpleasant hallucinations fail to depress the patient. The hallucinations or the train of thought and heightened feeling soon lead to delusions of an exalted character. The patient is a noble, or a king; is inspired, or is God; owns the earth; has supernatural powers; has hundreds of children and millions of money. But the flow of thought is too rapid for one delusion to be elaborated or brought logically into relation with surroundings; hence the delusions are many, are not fixed, and vary from day to day. Consciousness is apparently clear, but attention is so scattered that memory of what occurs in mania is very imperfect.

The outcome of the confused deluded thought is largely in insane action in mania, and acts of frenzy are frequent. Such acts may also be the result of direct cerebral excitement without any motive. The patient is in constant motion; shouts and screams; talks constantly, often in disconnected phrases, the rapid thought outrunning slower speech; tears his clothes off; acts without sense of decency; becomes in conduct a mere beast without restraint; gets into terrible rage with those about him, and becomes abusive, violent, unclean, and dangerous. Sexual excitement is often intense (nymphomania). The physical symptoms are few. All the bodily functions are, as a rule, well performed, though the constant activity leads invariably to great loss of weight and sleeplessness. Secretions are increased, as may be noticed by constant expectoration and frequent micturition. The patients appear to disregard cold and offensive odors, but are not anæsthetic. Temperature is not raised, and pulse is rarely increased in frequency. In states of frenzy great exhaustion may result.

The course of the disease is as follows: A gradual onset, with prodromic period of melancholia, and final development of all the symptoms of simple mania, which usually goes on to a frenzied state

with delusions; a stationary period of about three months and then a gradual recovery, whose beginning is shown by brief lucid intervals, in eighty per cent. of the cases. Death from exhaustion occurs in five per cent., and fifteen per cent. go on to chronic mania and terminate in dementia. The cases rarely last over six months—severe ones rarely over a year, and the intensity of the frenzy does not determine the duration. A recurrence is not frequent.

The treatment consists in isolation, and in case of violence it is far better to allow the patient freedom of movement in a warm, unfurnished room, under supervision of attendants, than to restrain him by the camisole. There is little danger of suicide. Good food and water must be given freely to keep up the nutrition and excretions—especially in puerperal mania. Long-continued warm baths and wet packs have a quieting effect and should be given daily. To secure sleep chloral or sulphonal, or bromide with ergot, or hypodermic injections of hyoscyamia $\frac{1}{100}$ gr. may be used. Morphine always excites a violent maniac unless given in dangerous doses. In the stage of convalescence small doses of opium may be used.

Dementia.

Dementia is a perverted intellectual state in which the normal processes of thought, reason, and volition are interfered with or suspended.

In the normal mind perceptions and ideas start up processes of thought along accustomed or logical lines of association, which reason guides to right conclusions and conduct by rejecting associations of ideas which are merely accidental and illogical.

In disease the normal train of thought may be disturbed in various ways.

(1) The perceptions and ideas may be wholly of internal origin and so numerous and disconnected that thought is confused, no natural association of ideas being possible between the subjects brought before consciousness. The mind then reacts to the various subjects before it without logical sequence. This condition is known as *confusional insanity.*

(2) The process of thought may be suspended by disease, so that perceptions and ideas fail to start associations, and voluntary logical mental action is arrested; all resulting emotion and conduct is then wanting, and the patient becomes a mere breathing automaton

Best to feed wild maniac thrgh. soft rubber catheter through the nose.

Ice-cap to head.
Excitement is due to cerebral congestion
Thence whenever possible use the
warm foot b.

without perception, ideation, emotion, action, or consciousness. This condition is known as *stuporous insanity.*

(3) In all the forms of insanity there are permanent cases which show no tendency to recovery. Thus, some patients with melancholia or mania continue to suffer from their delusions, which, becoming fixed and constant, hamper all their mental action and prevent logical thought and proper choice of conduct. Such imperfect mental action long continued leads to a permanent decay of mental power. Or confusional insanity, if not recovered from, leads in time to a total suspension of all logical processes and to a permanent decay of mental power. Or stuporous insanity, whether relieved by automatic acts or not, may continue, leaving the mind in a state of permanent decay. And lastly, all forms of diffuse organic brain disease, such as meningo-encephalitis (the lesion of general paresis), multiple sclerosis, or cerebral syphilis, which suspend the activity of the cortex or of its association fibres will produce permanent mental decay. Any of these diseases may therefore result in *terminal dementia.*

It is evident, therefore, that the condition of dementia, in which the normal processes of thought, reason, and volition are interfered with or suspended, may arise in various ways, however uniform its final result. The varieties of insanity included in dementia present therefore, different symptoms in their course.

1. Confusional Insanity (Amentia. Verwirrtheit. Wahnsinn) *is an impairment of mental action with confusion of ideas due to the presence of illusions and hallucinations, but without continuous uniform emotional disturbance or suspension of mental action.*

It begins suddenly in consequence of an exhausted physical condition after fevers, rheumatism, or great excesses, and occasionally after an emotional shock or a short period of mental depression. The patient suffers from headache, vertigo, nervous irritability, and insomnia, becomes conscious of difficulty in controlling his flow of thought, and then is startled by the appearance of illusions and hallucinations in all the senses, without order, and in rapid succession, which confuse his mind. The state resembles the delirium of fever in respect of the variety and illogical kind of thought. The patient is excited or depressed alternately by the hallucinations or is merely confused. Soon delusions based upon the hallucinations develop, but these are transient, and vary from hour to hour in character. The patient may think that he has sinned, or that he is being poisoned, or that he is persecuted, or that he is inspired, or that he is

wealthy. His acts depend upon the delusion present, are impulsive, without plan or purpose, and vary with his emotional state. He cries or laughs, is angry or merry, is fearful or jubilant, is noisy or quiet. No one emotion, action, or delusion is long continued. The consciousness is much clouded, the patient does not know his surroundings or family, mistakes strangers for relatives, cannot fix his attention continuously on anything, becomes easily distracted from any line of thought, is incoherent in his speech, sentences being incomplete and disconnected, and has little or no memory of what has occurred during his disease. The confusion of thought is partly due to weakness of mental action and partly to the many illusions which crowd upon the mind. Insomnia, persistent refusal of food, and consequent emaciation and exhaustion are common, and great prostration is felt during the lucid intervals when such occur. At times the patient may sink for some hours into a state of stupor. The duration varies from a few weeks (especially after fevers) to several months (especially after childbirth), and a gradual recovery occurs in seventy per cent. of the cases; the remainder going on to chronic dementia, or terminating by death from exhaustion.

The treatment resembles that for melancholia.

2. Stuporous Insanity (Primary Dementia) *is an impairment and suspension of psychical action in all its phases; perception, ideation, emotion, action, and consciousness being equally involved.*

It begins either gradually after great exhaustion from severe diseases or excesses, or suddenly after hemorrhage or emotional shock; usually in persons below the age of thirty, who are predisposed to nervous prostration by a weak constitution.

The patient sinks into a state of indifference and becomes slow in thought, stupid, sleeps easily, loses all spontaneity, and acts as if in a semi-stupor. He takes no notice of what is going on about him, and has few sensory perceptions, having to be fed and kept clean like a baby. His mind appears to be a blank, no sign of thought being ascertainable. He is not depressed, but is wholly indifferent, nothing being able to excite his interest. His stupor differs from that occurring in melancholia in its origin—his mind is not fixed on one idea by an emotional state.

The total lack of perception, thought, or emotion removes all stimulus to action, and he remains motionless, the muscles being relaxed, the heart weak and rapid, respiration slow and superficial, the limbs cold, cyanotic, œdematous, reflexes diminished, automatic acts performed without notice, so that he is dirty and liable to bed-

sores. He is speechless and questions produce no response. He appears to have no consciousness, though the eyes may be open and an occasional tremor or sigh may show that he is still alive. The urine contains an excess of phosphates. In females menstruation is arrested.

The course is a remittent one, for hours or even days intervene in the course of the stupor when a slight mental activity can be aroused; this, however, is never normal, and rarely accompanied by any excitement. Where there is some excitement there may be a tendency to repeat certain phrases or acts or to pass into a state of ecstasy or catalepsy.* The stupor lasts from two to four months, and then gradually subsides in ninety per cent. of the cases. Ten per cent. die of bedsores, cystitis, or pneumonia.

The patient being speechless and stupid, it is sometimes difficult to distinguish this disease from melancholia with stupor. But in stuporous insanity the history of the onset differs from that in melancholia; the facial expression is that of stupor, not of mental distress; the patient sleeps much. The occasional remission of the stupor is not attended by evidences of despair and attempts at suicide, and the physical signs of great prostration are much more evident.

The treatment consists of rest in bed with attention to cleanliness, and maintenance of bodily warmth; forcible feeding frequently both by mouth and rectum; stimulation and tonics, both general, and directed to the heart and respiration. The necessity of such measures, even though they involve forcible feeding for weeks, is evident, since such a large per cent. of cases recover.

3. Terminal Dementia (Blödsinn, Chronic Mental Deterioration. Persistent Mental Instability) *is the final result of all those forms of insanity which become chronic and do not terminate by death in the early stages.*

In some cases it is reached quite soon, as for example, after severe maniacal frenzy. In others it does not appear for many years after the onset of the insanity, as for example in confusional insanity. And different forms of insanity present many variations in their course down to the terminal stage in dementia.

Whatever the form, however, there is a progressive loss of the highest mental attainments, of the finer appreciation of moral obligations and of social restraints; then the intellectual activity, keenness of thought, judgment, and reason, with the allied powers of

* This variety of stuporous insanity is called Katatonia.

memory and imagination, suffer. In a later stage perceptive power, attention, concentration, and association of ideas become enfeebled, and the emotional state becomes unstable. Finally the vacant stare, the immobile face, the relaxed posture, and the lack of appreciation of surroundings indicate the total suspension of thought. Thus there is a progressive dissolution of the mental powers, those of the highest level of evolution being the first to succumb, but finally all levels being involved until the individual loses all personality and self-consciousness and is reduced to a breathing automaton.

In the advance of such a dementia there are two classes of patients which present marked differences. Some are frequently excited. Others are usually apathetic. The first class have usually suffered originally from mania, from melancholia with agitation, or from confusional insanity. They are noisy, restless, and excited, but have no clear delusion or definite emotion as a basis for such excitement, no motive for their actions.

The second class have usually suffered from melancholia or stuporous insanity, or from general paresis. They are quiet, motionless, and destitute of thought and feeling, unaware of the need of food, and dirty. The physical state of persons with terminal dementia is one of fair nutrition, increase of weight, and good sleep. Trophic disturbances, however, are not infrequent, with resulting eruptions, bedsores, fractures of the bones, and hæmatoma of the ears due to slight irritation or to injury. Finally these patients die of exhaustion. Asylum care is the only possibility in such cases.

Psychoneuroses.

The forms of insanity thus far studied are such as may develop unexpectedly in a perfectly well person without a nervous constitution or any hereditary predisposition to mental disease. They are usually produced by some severe mental or emotional shock, by continued worry, or by some acute or exhausting physical disease. They tend to recovery in a very large majority of cases, after running a typical course, during which the contrast between the previous state of health and the present one of insanity is well recognized. There is no special tendency to relapse, and such derangements are not transmitted to the next generation. The mental disturbance must be attributed to a functional change in the brain, due to disordered nutrition or circulation, or to toxic agents, since no charac-

teristic lesions are found in the few fatal cases. These points of general resemblance warrant the classification of *melancholia*, *mania*, *confusional insanity*, and *stuporous insanity* together in a group which is termed the PSYCHONEUROSES.

PSYCHICAL DEGENERATION.

There are a certain number of persons in the community who differ from others in the fact that they possess a weak, unstable, irritable, nervous system from their birth. Their parents have been intemperate, nervous, or deranged, or they have had severe diseases in infancy. The weakness shows itself often during childhood, but especially at puberty, or when the responsibilities and endeavors of adult life put the nervous system to any strain. If the history of such a person is investigated it will be found that his vital functions are unduly sensitive to external injurious influences, so that slight causes produce severe effects, an indigestion causing convulsions, a little rise of temperature causing delirium. His cerebral functions are easily disturbed, he is emotional, hypersensitive, suffers pain more keenly than others, and is more ardent or impulsive. His vaso-motor system is unstable, he flushes and pales easily, is subject to palpitation, is quickly affected by alcohol, and suffers on slight provocation from headaches and vague neurasthenic symptoms. His mental activity is often of a highly unstable type. He is very easily excited, amused, offended, or depressed, is rarely calm, but usually sanguine or despondent. He is often peculiarly indifferent to things which should excite his emotion, does not respect or attend to his relatives, and indulges in strange antipathies to those who are his best friends. He is also peculiar in his modes of thought. He may be imaginative, original, often brilliant in certain directions, but seemingly incapable of acquiring ordinary forms of knowledge in ordinary ways. He is therefore stupid in some directions, incapable of deep, intense concentration, and perhaps at times exceedingly illogical in thought and unreliable in statement. He is usually impulsive, acts without proper consideration, is ungovernable, and persists in his own way in spite of reproof, often doing queer things without any apparent reason. In serious matters he is unstable, shows little decision of character, and no power of continuous effort. His self-consciousness may be extreme, all external occurrences being brought into some improba-

ble relation with himself. His self-control is, however, poor, and hence he is liable to indulge in bad habits, especially sexual, and to smoke and drink to excess. He may be odd in manner and in habits. In short there is a noticeable lack of harmony in his mental processes which makes him erratic and peculiar, and a general irritable weakness of the nervous system which makes him incapable of enduring any physical or mental strain.

It is in this class of the community that neurasthenia, hypochondria, hysteria, and traumatic neuroses develop when their appropriate causes act upon the nervous system.

It is of course possible for great strains to produce similar results in a nervous system originally normal, but this is uncommon, and neurotic persons are usually predisposed to neurosis from their birth. Such a person may go through life without much suffering and never show evidences of insanity. Others are always known as eccentric persons, not to be relied upon, liable to do or say odd things. Others still pass the border-line between eccentricity and insanity, and develop mental symptoms so marked and so typical that every one declares them to be insane.

When such a person becomes insane it is usually difficult to fix the date of the onset, for there is no sharp line between his ordinary condition and the mental derangement—the latter creeps upon him slowly. In such persons apparently trivial causes, such as the change of character which normally occurs at puberty, or during pregnancy, or at the menopause; or some particular nervous strain, such as overwork or incessant worry; or some seemingly slight disease or injury may produce very serious results. If such a person becomes insane the course of the disease is usually chronic, and very irregular, with lucid intervals and relapses, but without permanent recovery. It rarely, however, progresses to the state of dementia. These patients are liable to transmit their disease to their offspring.

It is evident therefore that the forms of insanity which appear in such persons will differ in many respects from those already studied. They differ so widely that they have been grouped in a class which is called PSYCHICAL DEGENERATION. In this class, as in the class of psychoneuroses, there are several types to be studied.

Periodical Insanity.

Persons with nervous constitution are by no means free from the liability to the development of the psychoneuroses. These usually tend to recovery, and rarely recur in a healthy person. But in a nervous individual they show a marked tendency to recur. Hence, in considering the insanities which present themselves in the physically degenerate, it is necessary to mention *periodical insanity*—a mental derangement of the form either of mania or of melancholia, or of an alternation of these two (circular insanity), which occurs in attacks at regular intervals from puberty throughout life, each interval being one of fair health, and each attack lasting only a few months. It is a remarkable fact that in each attack the hallucinations, illusions, imperative conceptions, delusions, and impulsive acts are identical in character in the same individual. The early attacks may be slight, and later ones severe. They are usually more sudden in onset and shorter in duration than the ordinary psychoneuroses, and during the course the personality is usually completely changed. During the interval the patient is sane, yet presents numerous symptoms of nervous weakness. The interval may be months or even years. Occasionally the attacks occur with each menstruation. They are to be treated as already described, but the prognosis in such cases is much worse than in the psychoneuroses.

Partial Degenerative Insanity.

In the class of nervous individuals who present the typical characteristics of psychical degeneration, there are some who are peculiar for their very marked lack of self-control. Self-control, whether in respect to emotion, thought, or action, is the highest function of the mind, and its deficiency is universally regarded as evidence of mental instability. A man's character is largely dependent upon his power of self-control, a power which enables him to reject present pleasure as inferior to future gain, to exalt self-sacrifice at the expense of selfishness, and to suppress such instinctive acts as rage, conceit, or animal passions and appetites. Sane men vary greatly in the relative degree of self-control, so that much latitude is to be allowed in judging of its degree. But there are some persons in whom its deficiency is so complete that they cannot be considered

as exercising even a minimum amount, and these persons must be considered insane. The deficiency may not be attended by any marked defect in reasoning power, and does not lead to such delusions as appear in paranoia. Nor is it usually accompanied by any defect in self-consciousness. It is an evidence of degeneration in the mental sphere in some part of its domain, rarely in its entirety, and hence is termed *partial degenerative insanity*. It presents itself as a defect in emotional control (folie raisonnante), or in moral control (moral insanity), or in voluntary conduct (impulsive insanity—imperative acts).

(1) *Emotional insanity* (folie raisonnante) occurs chiefly in females of hysterical temperament, and is often associated with uterine disease. The patient shows evidences of bad temper, constant depression, and discontented, irritable, moody action. She has little energy, little courage, little happiness, and suffers constantly from mental distress and physical pain. The continued strain leads to occasional outbursts of temper, and to acts of rage and fury, which imply a total lack of self-control. In calmer moments she claims that she was unable to prevent these actions in spite of a knowledge of their evil character, that she had to do wrong, to act foolishly; and hence fears insanity. She may have attacks of anxiety as in melancholia—especially at menses. The attacks of excitement recur for years. They may be allayed by the use of morphine, but the danger of its habitual use is great in this class of persons.

(2) *Moral insanity* occurs in both sexes, and develops early in life. The patient appears to lack the ethical sense and, though taught the moral import of conduct, cannot grasp or retain moral ideas, and so fails to exercise that self-control over action which is necessary to morality. The distinction between right and wrong fails to appeal to such persons, the term "ought" has with them no force. Family affection, social restraints, the laws of decency, of decorum, of custom, are all disregarded, and they have no regard for truth. As children, these patients are incorrigible liars, mischievous to the point of villany, cruel, obscene, and destructive, the trial of their family and detested by acquaintances. As adults, they drift into crime of all kinds, and become vagabonds. At the same time the intellectual faculties may be keen in some directions, so that the patient may conceal or defend his evil actions, or contrive to escape their punishment. Sexual precocity and perversion are frequent in such persons. Such moral perversion occasionally ap-

pears only after some attack of mental disease. It is commonly associated with attacks of epilepsy. The absence of all sense of guilt, or penitence, or remorse, is a striking fact in moral insanity. It is termed by Krafft-Ebing moral idiocy.

(3) *Impulsive insanity* is characterized by a lack of control over morbid impulses, so that the patient does impulsive acts without being able to arrest them.

The impulsive acts may be either the result of imperative ideas, or the outcome of a sudden impulse to action, both being primarily the products of irritation in a defective brain.

Imperative ideas are ideas which arise without recognized association with such force as to impel to action. The fear of being watched, the fear of walking in an open place, the question whether one has offended a friend, whether one has contracted some disease, may come upon the patient with such intensity as to lead to instant action, which is repeated again and again, no certainty being reached by the act (folie du doute). A patient of my own will return sixty or more times to feel whether she has turned out the gas before getting into bed. There may be an impulse to mathematical calculation, so that everything raises some problem, sets up a train of thought which must be gone through before anything else is done. Some patients incessantly ask questions without waiting for a reply.

Impulsive acts may be done without any clear reasoning process, the impulse coming suddenly, being perhaps resisted and then yielded to. Thus the impulse to steal, to drink, to burn, to kill, to jump off a car or from a height, may overcome all scruples and be carried out immediately, in spite of efforts to control it (kleptomania, dipsomania, pyromania, etc.). The impulse to suicide may lead to the act in spite of religious considerations and the natural love of life. These acts resemble the acts of post-hypnotic suggestion, which cannot be resisted. And these patients often show little sign of intellectual weakness.

It is evident that such cases can only be explained by the hypothesis that self-control is abnormally weakened, that a partial mental incapacity is present. This weakened state may be only temporary, especially at puberty or after great sexual excess, or it may manifest itself for years.

Paranoia.

Paranoia (Primäre Verrücktheit, Chronic Delusional Insanity, Monomania) *is a chronic mental derangement characterized by primary fixed systematized delusions which dominate, but do not impair, the intellectual processes.*—This form of insanity usually develops slowly in a person of nervous constitution, who is predisposed to insanity by an hereditary tendency, at the time of puberty or in youth, or in women at the menopause, and runs a chronic course.

Its chief characteristic is the development of a delusion. This is said to be *primary*, because it is not based upon any permanent emotional state or upon hallucinations, but seems to be a direct result of the disease. Patients in every land thus afflicted present the same kind of delusions. The delusion is said to be *fixed*, because it is constantly before the mind, is never forgotten, tinges every perception, leads to emotion and action, and dominates self-consciousness. The delusion is said to be *systematized*, because it is logically elaborated and brought into association with other thoughts and ideas, so that it causes a complete change of character and personality.

The delusion may be either a delusion of persecution or a delusion of grandeur. It usually has a basis in hypochondriacal notions. The natural limitations or the necessary restraints of a weak neurotic person lead to his falling behind his companions in the competition of life. His inferior power is the last thing which he will admit, and hence he seeks to explain his defeats by the hypothesis of hostile influences surrounding him. His friends have taken advantage of him, his family has suppressed him, strangers are imposing upon him. He brings into relation with this new idea many past occurrences which come to mind, and begins to look out for further evidences. The indifferent glance of a stranger in passing is noticed, and thought to be one of suspicion or of disdain. If two persons are seen talking he imagines that he is the subject of their conversation, or that one is pointing him out to the other for some bad purpose. The words of a sermon, or in a play, are supposed to be applied directly to him. And thus the self being ever prominent, the entire world appears to revolve about it. Each form of opposition to his efforts affords new proof of the enmity of those about him, and since so many persons appear to act with the same motive, it is natural to suppose that they are all in league against

him. In some cases the conspiracy is thought to be limited to the members of the patient's family, in others to a church ("the Jesuits"), or to a secret society ("Freemasons"), or to the government ("the police," "the administration"), or to some prominent organization ("the Academy of Sciences," "the doctors"); or, finally, it may be universal, including every one knowing him, and many who are not acquainted with him, but who have been induced to join by his enemies. All sorts of motives are ascribed to these enemies, hatred, malice, but especially envy. And the latter idea, once reached, leads soon to the delusion of grandeur. For, if he is thus persecuted it must be that he is important, that he has great powers which they dread, great wealth which they are unjustly depriving him of, great station which they prevent him from reaching, great spiritual insight which they wish to conceal from the world. It is but a step for him to persuade himself that he is really a noble, a king, a deity, the greatest of poets, or authors, or scientists, or inventors, the most remarkable man in the world. Hence follows a change of personality, it being impossible for such a highly gifted being to have been an ordinary nervous man.

During the development of these delusions it is common to discover that hallucinations and illusions are occurring. Voices speak to him, forms appear to him, influences reach him by touch and taste, electricity acts upon him in an unknown manner, but perceptibly, and all confirm in one way or another the delusion, being brought into some relation with it whatever they may be.

The delusion influences his emotion, which is one of depression when the conspiracy seems to threaten him, or of great excitement, indignation, fear, terror, despair, when his enemies are on the point of triumph. The emotion changes with the delusion, and as his importance becomes prominent to his mind he feels hopeful, cheerful, proud, elevated, happy.

The delusion influences his conduct, for he may seek to evade his enemies by secluding himself, or may take vengeance on them by killing them, or may in despair commit suicide. The hallucinations may influence his action, he may obey the commands which he hears. At times the mental distress may lead to wild, uncontrollable excitement which is dangerous to those about him. When, however, he becomes proud and happy he shows his contempt for the rest of mankind by lofty bearing, by affected action which he thinks appropriate to his new personality.

His self-consciousness and power of attention and memory ap-

pear to be unaffected throughout, and he will talk quite freely of his birthplace and family, though declaring in the next breath that he is a noble or God.

From time to time there may be remissions in the symptoms, so-called lucid intervals, when the delusion sinks into the background, or when he even speaks of it as an insane idea. But sooner or later it returns in full force, being taken up at the point where it was when the lucid interval began, and so continues throughout life. Krafft-Ebing affirms that in five hundred cases he never saw a permanent recovery.

The variety of delusions which are found in different individuals is very great, and hence patients suffering from paranoia are not so easily grouped together as patients suffering from melancholia or dementia.

There are patients who have delusions founded upon abnormal bodily sensations, such as the delusion that an animal is living in the stomach, that the veins are full of steam, that the skin is made of glass. These partake of the character of hypochondriacal delusions, since they are erroneous explanations of ordinary sensations.

There are patients whose delusions are purely persecutory, without any tendency to develop into delusions of grandeur. Such patients are in constant fear of being poisoned, and frequently have to be fed, as they refuse all food. (Vergiftungswahn.) Others fear arrest and conceal themselves from their friends as well as from their supposed enemies. Others imagine that wherever they go some one is watching them, and influencing their physical condition by electricity or by some occult means; they invent all sorts of instruments to protect themselves, or finally take vengeance on their enemies. (Verfolgungswahn.)

There are patients who develop delusions of grandeur from the very beginning of the disease. They interpret the notice which they think others take of them as evidence that they are remarkable for beauty or for grace, or for some great ability, and thus at once begin to dwell upon their own importance and elaborate a delusion of their mighty power or great wealth. (Grossenwahn.) They claim to have made great attainments, and their failure to show evidence of these does not shake their confidence.

There are patients whose delusions of persecution lead to such a state of excitement that they desire legal revenge or legal protection, and are constantly appealing to the courts and obtaining the arrest of their supposed enemies, until the repetition of such baseless

proceedings leads to the conclusion that they are insane. (Querulantenirrsein.)

There are patients who accuse themselves unjustly for many things, thinking that their subjective feelings are a punishment for their sins. Such self-accusatory delusions resemble those of melancholia, but are more fixed and elaborated.

There are patients who are easily impressed by what they hear or see about them, and copy the delusions of others. The so-called folie-à-deux, in which two members of a family show the same delusion, is to be explained by the weaker mind succumbing to the influence of the stronger. The epidemic insanities of the Middle Ages were of this character.

There are patients whose delusions are chiefly of a religious type; they have been religious enthusiasts, and all their interests have been limited to religious matters, when the occurrence of an hallucination leads to the delusion of inspiration. They find in the Bible passages which refer to themselves, they hear God speaking to them, they lie for hours in a state of religious ecstasy, and then declare that they have a mission, that they are sent to reform society, to preach the true religion, to save the race, to be the Messiah. (Examples: Joan of Arc, the Mahdi.) They become at last so offensive that they have to be sent to an asylum, where they remain under the firm conviction that they are martyrs.

There are patients whose delusions are erotic in character. They fall in love with some great person, prominent actress, or fashionable lady, and spend their time devising romantic situations, in following the object of their affections, finally persecuting him or her in many ways by attentions. Hallucinations occur which convince the patient that the affection is returned, and these lead to open acts, which in turn prove to others that confinement in an asylum is necessary, where a delusion of persecution not infrequently follows.

These various forms of delusion have led to many subdivisions of paranoia which need not be enumerated. They all have this in common, viz., a well-defined delusion, which is brought into relation with all the life and thought of the patient, and dominates his action; an intense egotistical feeling; a change of character, and even of personality.

A patient presenting these characteristics is a paranoiac, and may be assigned to a hypochondriacal, religious, erotic, or other class of paranoia, according to the arbitrary classification adopted.

Some writers affirm that the disease may be congenital, signs of it appearing in childhood; and they distinguish such patients by the term originäre or congenital paranoia.

The only proper treatment of a paranoiac is confinement in an asylum, that he may be kept from excitement and from harm, and that society may be sure of immunity from sudden dangerous attacks. During lucid intervals a degree of freedom may be allowed, but always under observation, and under such restrictions that immediate return to the asylum may be secured when the delusion reappears. For this is the kind of patient who is a constant menace to society, history showing that attempts at assassination of prominent persons are made quite uniformly by paranoiacs.

The forms of insanity thus far studied are without any known organic basis of cerebral disease. They are ascribed to changes in the circulation or in the nutrition of the brain, or to congenital peculiarities of structure which interfere with the normal function of the cerebral cortex. There are, however, no constant pathological changes present in the brains of patients dying from any of these forms of insanity to which the symptoms can be ascribed. It is not impossible that future investigations may reveal the existence of pathological states not now known. But up to the present time the forms of insanity thus far considered must be regarded as being *without organic lesions.*

Organic Insanity.

There are, however, some forms of insanity of which a pathological condition is known to be the cause. These therefore are termed *organic* insanities in distinction from those thus far studied. Insanity of this character may develop in a healthy person or in one who has a degenerate nervous system, under the excitement of certain physical causes. It runs a progressive course and during its duration it may be transmitted to one's offspring. Recovery may occur if the physical changes in the brain can be arrested, but this is seldom possible.

Organic insanity presents itself in several forms, all of which have many common and a few distinctive features. The mental changes in all are such as may be ascribed to a gradually increasing loss of function from destruction of the cortex, a progressive dementia. There is a dissolution of mind, beginning in the highest level, that of self-control, and gradually extending to all the func-

Symptoms almost identical w- general paresis.

tions. The removal of restraint at first leads to symptoms of mental excitement, but these are soon followed by symptoms of mental torpor, and the termination is one quite similar to that already studied as terminal dementia.

In the *insanity of chronic alcoholism* erotic fancies, numerous fearful hallucinations, and delusions of conjugal infidelity are the special symptoms associated with the progressive dementia.

In the *insanity of senility* undue suspicion of friends, lack of memory of recent events, and consequent impairment of judgment, great egotism, particular susceptibility to flattery, and delusions regarding financial matters, usually of poverty, or of conspiracy to obtain money, are characteristic.

In the *insanity of cerebral syphilis* the mental symptoms are those of emotional excitement or depression, and of intellectual failure, but delusions and hallucinations rarely occur. The physical symptoms of gross lesions are usually found in these cases.

The most common form of organic insanity is *paretic dementia.*

Paretic Dementia.

Paretic dementia (General paresis, General paralysis of the Insane, Dementia paralytica, Softening of the Brain) *is a chronic diffuse encephalitis, presenting mental, motor, vaso-motor, and sensory symptoms, with progressive course and fatal termination.*

It begins very gradually, so that many cases are overlooked and others are considered as neurasthenia or dyspepsia for some months, there being present some of the symptoms of one or both of these diseases. After a time the friends of the patient begin to notice that he is not acting naturally, that he is unusually irritable or excitable, is offended or delighted at slight provocation, is variable in his moods, and appears at times inattentive, forgetful, and careless of proprieties. Soon it is found that his affairs are in confusion, his accounts not properly kept, and that it is impossible to keep his attention concentrated upon important business or upon any train of thought, so that his conversation becomes noticeably fragmentary. He cannot do his work and yet resents any interference with it, maintaining that he is perfectly well. His memory for recent occurrences then begins to fail, he neglects engagements. He disregards obligations, sleeps in company, loses his temper and uses bad language at home or in public, neglects his family, and, though previ-

ously moral in conduct, indulges in alcoholic and sexual excesses. By this time it is noticed that his speech is a little thick, indistinct and hesitating, and a fine tremor of the tongue and lips and possibly of the hands may be detected. The affection is one of fine co-ordinated movements—a cortical ataxia, rather than gross paralysis. The pupils are often unequal, or contracted, or dilated, and react very slowly to light.

During this period of invasion headache and sleeplessness are sometimes prominent symptoms and a gradual loss of weight goes on. From the outset restlessness is noticeable and after a time it seems impossible for the patient to keep quiet, he wants to take all kinds of exercise, to walk for miles, to go about even when exhausted by fatigue. His intellectual powers are notably weakened, he is no longer attentive, logical, self-controlled, cautious, or careful in conduct, and the change of character becomes apparent both to his family and to his acquaintances. This period of invasion varies from one to three years, and may be interrupted by periods of subsidence of many of the symptoms. It usually goes on to the full development of the disease.

When paretic dementia is fully developed numerous symptoms present themselves—both mental and physical.

The power of sensory perception is not impaired, but the patient is often so preoccupied or indifferent that he does not notice fully or remember the subjects to which attention is directed. His mind passes rapidly from one thing to another and he does not perceive accurately anything. Thus a patient will forget that he has dined, or neglect to put on some of his clothes, will expose his person, or will commit openly small thefts, will make serious mistakes in his accounts or appointments, merely from a lack of careful perception and consequent impairment of memory. Illusions and hallucinations occur only toward the close of the disease, and are followed by a marked loss of power of sensory perception when the dementia becomes extreme.

The power of logical thought is impaired very early. The patient shows impaired judgment from the outset, takes little notice of important matters, dwells upon trivial things, acts without consideration, and indulges in extravagant schemes, reckless expenditures, and excesses of many kinds which his better judgment would condemn. He may become exceedingly immoral, all restraints of society, religion, and morality being wholly neglected. He becomes profane, obscene, and tipsy. He very frequently develops a delu-

Let suspected H. write you a lett
he will omit one or two letters in
spelling many of the words.

sion of grandeur, says that he never was so well in his life, believes that he is the strongest, the brightest, the most powerful, the most wealthy of men. And he acts at times in accordance with his delusion, undertaking the most extravagant and impossible schemes without any regard for his actual circumstances. Yet his delusions are not carried out fully in his actions and he mixes ordinary affairs with them in an incongruous manner. The delusion is not as fixed or as systematized as in paranoia. The development of such a delusion is a sure sign, in Meynert's opinion, that an atrophy of the brain has begun.

His emotional state is unstable. He has little control over its manifestations, can be moved to tears or to laughter in the course of an ordinary conversation, can be excited or depressed by suggestions. He is sometimes depressed for days, and feels discomfort, but does not blame himself, and in the midst of the depression may become excited. He is usually sanguine and hopeful, and his emotion is usually in accordance with his delusion, one of exaltation. At times or on provocation the excitement may become maniacal, and outbursts of rage or of frenzy are not infrequent in the course of the disease. Hence a paretic dement is never a harmless member of society, but requires to be watched. He rarely shows any tendency to suicide.

Voluntary action and conduct are affected from the outset; indeed it is by change in conduct that the changes in thought and character are chiefly betrayed, and the weak logical power made manifest. Inconsistency is evident in speech and conduct. Acts are done without consideration; thus impulsive acts, such as stealing, forgery, enormous expenditures, even murder may be performed under sudden excitement or under the influence of the delusion, or in a fit of frenzy. All the ordinary restraints to conduct seem to be removed, and instinct rather than morality is the guide, self-control being greatly weakened.

Consciousness appears to be very imperfect. The patient does not appreciate the inconsistency between his previous personality and his present acts; he does not notice the growing anxiety of his family; he regards with indifference loss of means, or even his confinement in an asylum; he is usually so engaged in his imaginary undertakings under the influence of his delusion that he is contented wherever he is. His personality may be changed, and he may imagine himself a prince, a millionaire, a deity. Gradually the consciousness becomes obscured, he recollects little regarding his

illness, and as the deeper dementia ensues he becomes indifferent, dull, stupid, and finally almost unconscious. In the final condition of dementia all mental action is suspended.

The physical symptoms are as marked as the mental symptoms.

Motor disturbances appear early. There is restlessness, a tendency to be in constant motion. The patient takes long walks or rides, indulges in unusual exercise, wishes to be going out to theatres every night, to visit friends all the time, and is not content to lead an ordinary quiet life, as before his illness. He has a sense of physical power and is eager to show his strength, though this may really be impaired. He is very talkative, discussing subjects of which he knows little—or talking at random. Tremor of the muscles appears early, first in the tongue, then in the face and hands, and finally any motion is attended with some trembling. This tremor is followed by inco-ordination, which shows itself in thickness and indistinctness of speech, in irregularity of handwriting, in clumsiness in handling things, and an awkward, unsteady, stumbling gait. The facial expression becomes blank and inane—no appearance of thought or interest being manifest, but when he talks an excessive play of facial expression is noticeable. Paresis soon shows itself, and progresses, until in the final stage there is total paralysis, with increased reflex action and loss of all control over bladder and rectum. Finally speech may become impossible.

In the course of the disease epileptiform attacks, attacks of sudden loss of consciousness without convulsions, and attacks of monoplegia or hemiplegia, temporary or permanent, often occur.

Vaso-motor disturbances are also present. The face flushes or pales frequently, there are sudden attacks of vertigo and of headache, and of feeling of fulness in the head; the pulse is usually slow, large, and of low tension. In the late stages venous congestions in various organs are found.

Sensory symptoms are less marked than motor symptoms, but as the disease advances anæsthesia or analgesia of the limbs may be found, especially in connection with hemiplegia.

In the last stage trophic disturbances occur, for in the stupid, dirty, helpless state of the patient cleanliness is difficult and the liability to bed sores and to cystitis is great.

Some cases are complicated by the development of spinal sclerosis, either in the form of posterior or of lateral sclerosis with its attendant symptoms, and in a few cases the paresis follows the spinal disease.

If epilepsy develops after 45, it is usually organic often points to beginning Paresis.

Syphilis of Brain	Paresis
Headache more prominent symptom & more common at night.	
Loss of faculty more pronounced	Excitement & faculty more pronounced
Sleeplessness more pronounced	
Attacks of paralysis or paraesthesia in limbs more common	
Irreg. of pupils more common.	More likely to be equal & contracted
	Tremor of muscles more likely here
Disease progresses in attacks.	Progress of dis. more rapid & continuous

It is evident that the mental and physical symptoms are very numerous. Many cases show only some of them; others present all the symptoms during the course. In some cases the mental symptoms appear early and are more prominent throughout than the physical symptoms; in other cases the reverse is observed. A few cases begin with epileptiform attacks. The course of the disease is slowly progressive. Its average duration is about three years, though rapid cases may terminate within a year, and some are known to have lasted five or six years. The patients die of exhaustion or of some complicating disease such as cystitis, or pneumonia, or obstruction of the bowels.

Pathology.—The disease begins with changes in the vessels, hyperæmia of the pia and small cortical vessels, increase of nuclei in the vessel walls, occasional stasis in the capillaries, and exudation of serum into the lymph spaces. As it increases, a formation of fusiform dilatations of the vessels and the development of fibrils of connective tissue between the vessel walls and the surrounding neuroglia occur. In the neuroglia at the same time a marked increase of the nucleated cells with numerous branching processes is in progress, numerous spider cells developing throughout the cortex, around the vessels and lymph spaces, and about the nerve cells. This increase of neuroglia goes on rapidly, producing a diffuse sclerosis of the cortex which is followed by retraction of the tissue, leading to atrophic shrinking. The entire brain takes part to a lesser degree in this process. In the cerebral tissue at the same time there is in progress a degeneration of the finest nerve fibrillæ and of the branching processes of the nerve cells; and also a swelling, hyaline or fatty degeneration, vacuolization, pigmentation, and final atrophy of the cell bodies, and the formation of cystic cavities throughout both gray and white matter.

The final result of these pathological processes in the vessels, neuroglia, and cerebral substance is a gradual atrophy of the brain, so that it weighs much less than normal, it appears shrunken, the convolutions being narrow and the sulci open; the brain tissue is hard, pale, friable, pigmented, and on the summit of the convolutions adherent to the pia, which is opaque and thickened. A thickened condition of the ependyma of the ventricles is uniformly found. There is usually an increase of fluid within the ventricles and beneath the pia. As a complication the lesions of pachymeningitis with hæmatoma are not infrequently present. All of the changes in the cortex are more marked in the frontal lobes and about the

Sylvian fissures. In many cases the lesions of posterior or of lateral sclerosis are found in the spinal cord.

Etiology.—The disease occurs between the ages of thirty-five and fifty-five, in males chiefly, after alcoholic or sexual excesses, great emotional shock, or severe mental strain, and in a large proportion of cases in syphilitic patients. Chronic pachymeningitis or multiple cerebral sclerosis may give rise to a very similar train of symptoms.

Treatment.—In the early stage general tonic treatment, combined with hydrotherapy and the continuous use of ergot, is recommended. In syphilitic cases antispecific treatment should be tried. For states of excitement, purgatives, bromide, chloral, paraldehyde or sulphonal, physostigma, or morphine may be used. The absolute abstinence from alcohol should be enforced. In the later stages nothing can arrest the disease, and home or asylum care is necessary.

Classification of Mental Diseases.

A. Mental Diseases without Organic Lesions.
- I. Psychoneuroses, in healthy brain.
  - 1. Melancholia.
    - *a*, simple.
    - *b*, with delusions.
    - *c*, with agitation.
    - *d*, with stupor.
  - 2. Mania.
    - *a*, simple.
    - *b*, with frenzy.
  - 3. Confusional Insanity.
  - 4. Stuporous Insanity.
    - *a*, simple. Primary dementia.
    - *b*, with periods of ecstasy. Katatonia.
  - 5. Terminal Dementia.
- II. Psychical Degeneration, in weak brain.
  - 1. Periodical Psychoneuroses.
  - 2. Partial Degenerative Insanity.
    - *a*, emotional. Hysterical.
    - *b*, moral.
    - *c*, impulsive.
  - 3. Paranoia.

B. Mental Diseases with Organic Lesions.
   1. Alcoholic Insanity.
   2. Senile Dementia.
   3. Syphilitic Dementia.
   4. Paretic Dementia.

C. Mental Defects with Maldeveloped Brain.
   1. Imbecility.
   2. Idiocy.

The Causes of Insanity.

Insanity is rarely if ever due to a single cause; it is usually the effect of a series of causes acting through a considerable period of time and brought to a culmination by some comparatively insignificant event. It is therefore necessary to consider both the predisposing and exciting causes of insanity.

The predisposing causes of insanity may be divided into two groups, the general tendencies which affect the race and the special tendencies which affect particular individuals.

Among the *general tendencies* the most potent is the struggle for existence incident upon the increasing competition in civilized communities. As a country becomes more densely populated, as people gather in cities, as interests widen and responsibilities grow heavy, as the distracting elements of life increase, as greater effort and more intense energy become necessary to success, and as temptations to vicious indulgence multiply, so the ratio of insane to sane steadily rises. In the United States the ratio was 1 to 1,310 in 1860, 1 to 1,030 in 1870, and 1 in 545 in 1880, and in New York State 1 to 374 in 1890. In cities the ratio is much higher than in the country. Out of 16,000 insane in New York State in 1890, 7,500 were in New York city and Brooklyn asylums.

Race and climate appear to have little to do with the development of insanity. This is true in spite of the fact that in the United States nearly one-third of the insane are foreigners, though but one-seventh of the population is foreign. The sexes are about equally liable, though the census of 1880 shows that the ratio of men to women insane in this country was as 100 to 109. Insanity is a disease of adult life, by far the larger number of cases occurring between twenty-five and thirty-five years in women, and between thirty-five and fifty in men.

Among the *special tendencies* to insanity affecting individuals heredity must be considered the most potent. In nearly three-quarters of all the insane cases observed the world over some hereditary taint can be discovered. This hereditary tendency may be derived from near or distant relatives, and the disease in the ancestor may have been insanity, or some nervous disease of organic or of functional, or of toxic (alcoholic) origin.

Next to heredity comes a neuropathic constitution as a predisposing cause of insanity. This has been already noticed in discussing psychical degeneration. An unduly sensitive or a weak nervous system succumbs more easily to the exciting causes of insanity than a strong one, and is subjected to undue strain even by the physiological changes occurring at periods during life.

Another predisposing cause is improper education and training. The urging to overwork which produces "precocious children," the foolish indulgence which produces "spoiled children," the deficient tact and cruelty which produces "repressed children," all imperfect educational methods which hinder an harmonious development of mental traits and fail to develop character act as predisposing causes to insanity.

Lastly, the occurrence of one attack of mental disease predisposes to a second attack.

The *exciting causes of insanity* are partly psychical and partly physical. Emotional shock, intense grief or anxiety, sudden joy, continued worry, undue sympathy, or long-continued intense intellectual effort kept up under a strain are the most common psychical causes. Severe exhausting diseases of any kind, injuries and inflammatory diseases of the brain, continued neuroses, especially epilepsy, sexual excesses, chiefly masturbation, the nervous strain of childbirth and the menopause, sunstroke, the abuse of alcohol, opium, or other drugs, are the most common physical causes. There are but few cases of acute insanity in which a history cannot be obtained of some illness impairing the strength and producing anæmia. In a small proportion of the cases, however, no cause can be ascertained.

The following were the chief causes assigned for insanity out of 1,400 cases admitted to New York State asylums during 1890:

Alcoholism, 253; Emotional strain, 247; Ill health, 163; Masturbation, 81; Old age, 80; Overwork, 78; Epilepsy, 74; Heredity, 59; Childbirth, 42; Traumatism, 41; Menopause, 38; Sunstroke, 29; Syphilis, 22; Abuse of Drugs, 22.—*Second Report State Com. in Lunacy.*

The Prognosis of Insanity.

The popular idea that the insane are incurable is erroneous.

By far the larger number of those who become insane suffer from the psychoneuroses, and in these forms of insanity over sixty per cent. recover. The incurable cases belong to the class of psychical degenerations, but, even in these, lucid intervals of apparent recovery of some duration may occur. The organic insanities rarely recover, and do not present long intermissions, but go on rapidly to death.

The longer the disease lasts the less likely is it to pass away. Thus, of the cases which recover, sixty per cent. get well within the first six months and twenty-five per cent. within the second six months. There is always hope until dementia appears.

The more sudden the onset of the insanity the better the prognosis. Thus the insanity of childbirth usually passes away.

The younger the patient the better the prognosis. Thus a melancholia developing in old age has a bad prognosis.

The symptoms which should make one dread an incurable course are persistent mental confusion with loss of memory, disregard for decency, impulsive acts, and the development of fixed and systematized delusions; convulsions or paralysis, and a great improvement in weight and physical condition unattended by mental recovery.

If proper precautions are taken to avoid exposure to the causes which have produced the first attack, a second attack may be averted, but asylum statistics show that a quarter of the patients who are discharged recovered return insane after a time.

Those who have been insane should not be allowed to marry, unless the insanity was of the form of a psychoneurosis and five years have elapsed without a return.

The Diagnosis of Insanity.

Difficulties in the diagnosis of insanity usually disappear as the knowledge of a case becomes more complete, and if all the facts, both positive and negative, are ascertainable, a conclusion should not be difficult in any case.

In the investigation of a person whose sanity is suspected, it is necessary to obtain a full history of the patient before seeing him,

and to ascertain what particular evidences of a change of disposition or of character have been noticed by his friends. In obtaining this history the following facts should be elicited:

(1) The hereditary tendencies of the patient.

(2) His life history up to the time when insanity was suspected.

(*a*) During childhood were nervous symptoms noticed?

(*b*) At puberty were eccentricities remarked?

(*c*) In adult life were his keenness of perception, intelligence, and character, disposition and self-control, occupation, habits, and actions those of a normal person, the relative social, intellectual, and moral status of the individual being considered?

(3) The psychical or physical events which have been thought to lead up to the present state. To what stress has he been exposed?

(4) What evidences in his appearance, manner, habits, or actions have led to a suspicion of insanity?

These facts must be obtained from relatives or friends, and will determine whether an examination is warranted, and will give a clue to the form of insanity present, to the particular points to be ascertained at the examination, and to the manner of proceeding to that examination.

For from these facts it will be evident whether the patient is undoubtedly insane, or probably insane, or possibly insane; whether direct approach and direct questioning as a physician will enable you to detect his mental symptoms better than to make his acquaintance without his knowledge that he is being examined.

In every case where it is possible the physician should examine the patient as a physician, so as to be able to ask such questions as only a physician is warranted in asking.

The examiner should observe the facial expression, posture, manner of action, dress, character of speech, handwriting, and physical power of the patient, and during the conversation notice the mental capacity as displayed by his memory of events, continuity and activity of thought, logical flow of ideas, and emotional state. It is better to question him regarding physical symptoms and during this investigation to estimate the mental condition. For a discussion of the reasons for the insomnia, or dyspepsia, or palpitation, or pain, or disturbance of sensation complained of frequently elicits explanations which betray the existence of delusions or of hallucinations, and a little display of curiosity as to these will lead to their full recital. Never show any knowledge of a patient's delusion

excepting such as he himself gives you; let him talk about it without interruption, if he will; and never let him suppose that you doubt his statements or suspect the possibility of his explanation of facts. The delusion is a real belief to him, and incredulity on your part merely awakens his hostility. A thorough medical examination alone may so command his respect and awaken his confidence that he will open the subject which is before his mind. If not, some discussion of his occupations or plans, of his family relations and business interests, of the possibility of a vacation or of travel may lead up to the delusion, or may betray peculiar lines of thought and views of life. It is well to talk with a patient both alone and in the presence of members of his family to observe whether his relations with them are natural.

There is never any difficulty in eliciting the symptoms of the psychoneuroses by such a direct examination, and their diagnosis is easy. In many cases of psychical degeneration, especially in paranoia with delusions of persecution, the patient will allude at once to his enemies and their designs upon him. In many cases of paretic dementia the delusions of grandeur will be easily reached from the boastful assertions regarding the patient's health. And in all forms of dementia the mental incapacity will be apparent.

In the examination it is well to bear in mind that the condition of (*a*) conscious perception, (*b*) logical thought, (*c*) emotional control, (*d*) volition, conduct, and self-control, and (*e*) personality are each to be investigated, all possible disturbances being watched for; and that in almost every case of insanity physical symptoms are present as well as mental changes.

The chief difference between a physical and a mental examination is that in the latter an estimate must be reached by an indirect method, for no man's statements regarding his own mental processes are to be accepted. The mental process must be judged by the observation of his mind in action. To warrant a diagnosis of insanity marked quantitative or distinct qualitative changes in previous mental action must be ascertained. Thus demonstrative changes of mood in a reserved man mean more than in an hysterical woman, and unusual wit in a dull man or extravagance in a miser are suspicious. The admission of hallucinations, delusions, or morbid impulses is very important.

There are some patients in the early stages of paranoia or of paretic dementia who are aware of the fact that their conduct has awakened suspicion, and who are on their guard under any examina-

tion, and careful not to betray any evidences of insanity. They are fearful of commitment to an asylum, and they refuse to see a physician, either maintaining that they are perfectly well, or asserting that their anxious friends are their worst enemies, and that the physician is in league with them. It may be evident from the history that the patient has symptoms of insanity, or it may be possible that his actions are those of a sane man who is really being persecuted. Yet in such cases it may be necessary for the patient's good to determine the question of his sanity, in order to prevent reckless extravagance, or in order to protect individuals from unjust or violent accusations, or in order to guard against suicide. Under these circumstances a meeting between the patient and the physician may be brought about by friends, and thus, without revealing his identity, the physician may estimate the patient's mental condition. Such a method is attended by difficulties, and is not always successful, and requires much tact and skill, that the real motive may be concealed. For a man justly resents such an attempt to deceive him, and the circumstances prevent the physician from entering upon many subjects which he may desire to know.

If such an attempt is made the physician should be in possession of all the facts regarding the patient's mental condition, so as to so adapt his method of procedure to the patient's personality as to elicit the necessary evidence of his delusion, or of his change of character, and several attempts may be necessary before a conclusion can be reached. It is necessary to refuse to make a diagnosis, or to be party to a commitment, when any interpretation of the history and of the facts observed leaves a doubt in the mind as to the insanity of the indvividual. And it is always to be remembered that a harmony between the facts of the history and the facts observed must be established before an opinion can be reached. Thus a history of melancholia and the signs of paretic dementia cannot be harmonized, if the examination points to paranoia, but investigation fails to reveal any history of psychical degeneration; or, if there is a history of degeneration but the observation reveals no physical or mental signs of it, a lack of harmony is evident. It is to be remembered that one symptom is insufficient to warrant a diagnosis, and that insanity has a typical course and is a nervous disease with adequate causes.

After the facts and the history have been fully ascertained, the diagnosis as to the form of insanity present is not often difficult when the characteristic features of the various forms are remembered.

Always keep a history of a lunatic - to ke
yourself protected from imprisonment

There are some persons who desire to be considered insane in order that they may escape punishment for crime. The simulation of insanity is difficult, for there must be in real insanity a harmony between the many disturbances of mental action and a typical course. In any suspected case long-continued watching will usually produce evidences of deception.

There are some criminals who are manifestly insane. Thus (1) when a man is incapable of distinguishing right from wrong in reference to a particular act, as in idiocy, imbecility, dementia, acute mania; or (2) when a man is acting under an insane delusion as to circumstances which if true would relieve the act of responsibility, or where his reasoning powers are so depraved as to make the commission of the particular act the natural consequence of the delusion, as in chronic mania or melancholia, or in paranoia; or (3) when a man is forced by a morbid and irresistible impulse to do a particular act, as in psychical degenerative states—the law allows the plea of insanity.

There are other cases which are doubtful. But when a man has a bad heredity, has been exposed to exciting causes of insanity, has developed an acute psychoneurosis and recovered, or has been eccentric or deficient mentally, or has shown previous symptoms of mental disease and at the time of the crime presents other symptoms and physical signs, and when the act of which he is accused was without motive, was not conducted with ordinary care and not followed by ordinary precautions to escape detection, or was either freely confessed or utterly denied with apparent ignorance, and gives rise to an honest remorse, there is a presumption in favor of his insanity. But if a man is acquitted of crime because he is insane, the law ought to enforce his immediate confinement in an asylum.

Asylum Commitment.

Patients can be committed to an asylum in New York State only by some legal representative (relative or guardian) after an examination by physicians who have qualified before a Judge of a Court of Record as Commissioners in Lunacy. Any physician can so qualify after he has been in practice three years by appearing before the Judge with two certificates from two Commissioners, known to the judge, certifying to his capacity. His certificate must be filed with the State Commission in Lunacy.

In order to commit a patient two Commissioners in Lunacy must examine him, independently or together, and must make out a certificate upon a legal blank furnished by the State Commission in Lunacy, in which certificate it is necessary to give full particulars regarding the case, stating the date of the attack, whether it was sudden or gradual in onset, the bodily condition of the patient, whether he is epileptic, whether he is filthy or cleanly in dress and appearance, whether he is violent, dangerous, destructive, excited or depressed, homicidal or suicidal, what is the supposed cause, whether he has insane relatives, what his habits are, what he said and did before the examiners, what his appearance and manner were, and what facts bearing upon his insanity were ascertained.

This certificate is signed and sworn to by the two physicians together before a Judge of a Court of Record of the county in which the lunatic resides, and the Judge may take other proof or make inquiry before approving the certificate, or may call a jury to decide the question of lunacy.

When the certificate is signed by the Judge, the patient may be removed to the asylum by his friends, who have a right to call upon the police if needed to assist in the removal.

When the patient has recovered he can be discharged by the head of the asylum. He can be transferred to another asylum by request of his friends at any time during his illness without new papers. If he escapes and is gone a month, new papers are necessary for his recommitment.

Except in mild melancholia - institution
care is best.

Peterson on Insanity.
Kirchhoff's Handbook of Insanity. Am. Text.
Manual of Insanity. Savage.
Krafft-Ebing of Insanity in German - Lehrbuch
der Psychiatrie.